Comprehensive Imaging Review of Abnormalities of the Placenta

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Abstract: The placenta has a fundamental role in fetal health and functions as an important bridge to normal fetal development throughout pregnancy. A complete fetal ultrasound (US) survey should include full assessment of the placenta for any possible abnormalities. Placental diseases range from abnormal morphology, size, location, extent, and degree of placentation, to abruption and the presence of rare placental neoplasms of benign or malignant nature. Some of these conditions are associated with other diseases including aneuploidies, and their discovery should alert the radiologist to perform a very thorough fetal US examination. At times, a fetal karyotype may be needed to provide additional information. Timely detection of placental abnormalities can alert the clinician regarding the need to make important management decisions to reduce fetal and maternal morbidity and mortality. Familiarity with the normal and abnormal imaging appearance of the placenta is therefore necessary for the radiologist. Ultrasound with Doppler is the initial imaging modality of choice for placental assessment. Magnetic resonance imaging serves as a problem-solving examination in instances where the US findings are equivocal or where additional information is needed. Computed tomography has a limited role in the evaluation of placental disease because of its relatively limited tissue characterization and in particular because of the resultant direct radiation exposure of the fetus. However, in specific instances, particularly after trauma, computed tomography can provide invaluable information for patient management.

Key Words: placenta, fetal, placentation abnormality, abruption, gestational trophoblastic disease, placental imaging, placental tumors, placental abnormalities

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The placenta plays a crucial role in normal fetal health and development. Many abnormalities of the placenta herald the possibility of future fetal compromise and, therefore, can be regarded as an "early warning sign." Placental development begins at the time of blastocyst implantation, and throughout the pregnancy, it serves as an "all-in-one" respiratory, excretory, and endocrine organ, while providing nutrition and immune support for the developing fetus.[1] Placental dysfunction during pregnancy can result in pregnancy-induced hypertension, fetal growth restriction, or fetal loss.[2] Compromise of fetoplacental circulation is suspected in approximately 20% of stillbirths at the time of autopsy.[3]

In this article, we will review the development, normal imaging appearance, and pathological conditions of the placenta. We will also recommend an algorithmic approach for assessment of the placenta, highlighting the active role of the radiologist in management of pregnancies with placental abnormalities.

PLACENTAL DEVELOPMENT

The placenta is anatomically divided into fetal and maternal portions. The fetal portion originates from the blastocyst, which implants in the endometrium on days 5 to 6 after fertilization. The portion of the decidua intervening between the blastocyst and the uterine wall is named the decidua basalis or the decidua placentalis; it is here that the placenta is subsequently developed.[4]

On weeks 3 to 4 after fertilization (weeks 5 to 6 gestation), the outer cell layer of the blastocyst transforms to the trophoblastic cell mass, which intermixes with the endometrial cells. The trophoblasts shortly thereafter differentiate into the cytotrophoblast and syncytiotrophoblast. The syncytiotrophoblasts form numerous processes, namely, the chorionic villi, which invade and destroy the uterine decidua. The chorionic villi are subsequently vascularized by growth of the branches of the umbilical vessels into them.[2] The decidual lining forms the maternal portion of the placenta. Before true maternal blood flow is established at week 10 after fertilization (12 weeks of gestation), plasma flows into the intervillous spaces. Uteroplacental circulation is established when uterine spiral arterioles are transformed into low-pressure, low-impedance vessels. Fetal blood is then carried to the villi by the branches of the umbilical arteries and, after circulating through the capillaries of the villi, is returned to the embryo by the umbilical veins. Overall, there is a decrease in resistance to blood flow in the uterine circulation from early gestation to term in normal pregnancies.[5]

The placenta therefore has 2 circulatory systems: the maternal-placental (uteroplacental) and the fetal-placental (fetoplacental). The former allows flow of maternal blood into the intervillous spaces via the decidual spiral arteries resulting in exchange of oxygen and nutrients, whereas the latter allows exchange of the deoxygenated and nutrient-depleted fetal blood with maternal resources in the terminal villi (Fig. 1).[6]

The placenta maintains its proliferative nature during the first half of the pregnancy. In the second half of the pregnancy,

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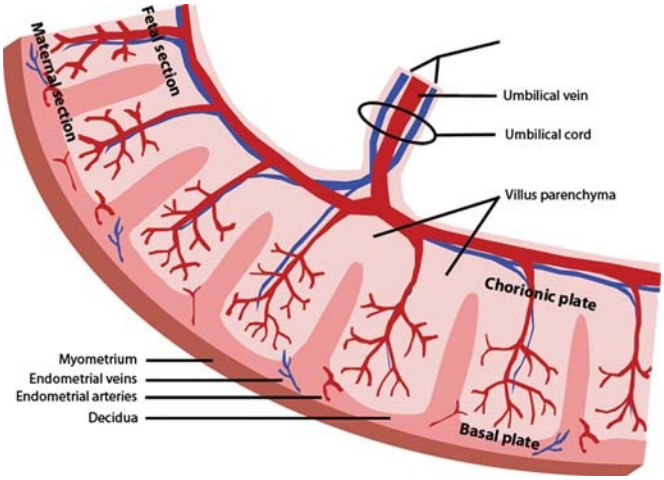

FIGURE 1. Diagram depicting a cross-sectional of the placenta and umbilical cord. Note vascularized chorionic villi communicating with small vascular branches arising from the basal plate.

the placental growth decreases while its maturation rate continues to progress.[2] At term, the maternal blood flow to the placenta is at about 600 to 700 mL/min.

IMAGING METHODS

Ultrasound (US) is the initial imaging modality of choice because of its safety in pregnancy and availability and the ready visibility of the placenta.[7] Magnetic resonance imaging (MRI), with its high spatial resolution, multiplanar capabilities, large field of view, and high soft-tissue contrast, usually serves as a problem-solving examination. Magnetic resonance imaging can demonstrate the uterine wall, placenta, amniotic fluid, and fetus, all with excellent tissue differentiation.[8] Computed tomography can be used for evaluation of trauma to the maternal abdomen and pelvis, to the placenta, and to the fetus, as well as for the evaluation of metastases associated with gestational trophoblastic disease.[9,10]

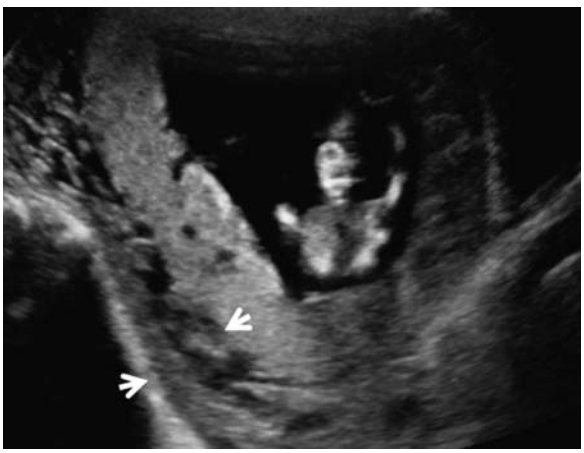

FIGURE 2. Normal placenta at 12-week gestation. Sagittal gray scale ultrasound image shows the hypoechoic ''retroplacental complex'' (short arrows).

ULTRASOUND

On US, the placenta is visible as early as 6 weeks of gestation with transvaginal US and by 10 weeks of gestation with transabdominal US. It appears as a hyperechoic thin rim around the gestational sac.[11] Intervillous blood flow is demonstrated by Doppler US by 12 to 13 weeks of gestation. By 14 to 15 weeks of gestation, the placenta is well formed and appears as a prominent hyperechoic area.[12] The "retroplacental complex," composed of the decidua, myometrium, and uterine vessels, is also readily seen (Fig. 2). With increasing maturation and size in the second trimester, the placenta appears more homogeneous and hyperechoic (Fig. 3).[13] It may contain internal ill-defined hypoechoic areas representing placental lakes. By the third trimester, the placenta appears as a highly vascular organ on Doppler imaging. The normal placenta at this stage of gestation has a rounded margin with a thin or sheet-like edge.[13]

Ultrasound evaluation of the placenta includes assessment of size, thickness, and echotexture. Normal placenta at term measures 15 to 20 cm in diameter and ranges from 2 to 4 cm in thickness (Fig. 3).[14] Hoddick et al reported that placenta grows in thickness throughout pregnancy and should measure approximately the same as the weeks of gestation in millimeters; however, it should not exceed 4 cm.[15]

Placental changes during pregnancy can be correlated with fetal maturity. As the placenta matures, it develops calcifications. This feature is used in placental grading and can be helpful in prediction of adverse fetal outcomes.[7] The placenta grading developed by Grannum et al on US is as follows[16]:

Grade 0: Late first trimester to early second trimester (10–17 weeks); uniform moderate echogenicity; smooth chorionic plate without indentations.

Grade 1: Mid second trimester to early third trimester (18–29 weeks); subtle indentations of the chorionic plate; small, diffuse calcifications randomly dispersed in the placenta.

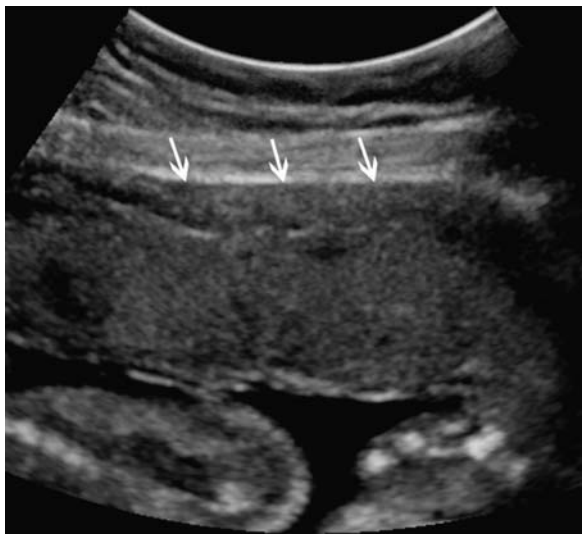

FIGURE 3. Normal placenta at 24-week gestation. Transverse gray-scale ultrasound image shows normal placental echogenicity, thickness, and the relatively hypoechoic retroplacental myometrium (arrows).

Grade 2: Late third trimester (30 weeks to delivery); larger indentations along the chorionic plate; larger calcifications in a "dot-dash" configuration along the basilar plate (Fig. 4).
Grade 3: (39 weeks to post dates); complete indentations of the chorionic plate through to the basilar plate creating "cotyledons," which are portions of the placenta separated by these indentations; more irregular calcifications with substantial shadowing.

A lack of progression through the various placental grades/maturation process seems to have no clinical significance; however, preterm placental calcification/premature calcification increases the risk of adverse fetal outcomes, such as preterm birth, low birth weight, low Apgar scores, and neonatal death, and maternal adverse outcome, including postpartum hemorrhage, placental abruption, and the need for maternal ICU care. This calcification presumably reflects underlying insufficiency of the placental vascular supply (Fig. 4).[17] Hopper et al, in a study of 1096 pregnancies, found a correlation between early maturity of the placenta and adverse fetal outcomes. These authors recommended close clinical and US follow-up if a grade I placenta is noted before 27 weeks, a grade II placenta is noted before 32 weeks, and a grade III placenta is seen before 34 weeks of gestation.[18]

The introduction of 3-dimensional (3D) US has permitted a new approach to the assessment of the placenta. 3D surface-rendered imaging provides more detailed information on abnormalities involving the curvature and continuity of the placenta and the spatial relationship of placenta accreta and serves as a more easily comprehensible visual tool for the referring physician and the parents.[2]

Advances in 3D US technology have made it possible to assess the placental volume and its vascular status using power Doppler.[19] Three-dimensional placental volume measurement is done using traditional multiplanar and rotational techniques. Newer techniques including virtual organ computer-aided analysis and extended imaging virtual organ computer-aided analysis methods have also been reported in the literature. Details of these are beyond the scope of this article; however, both require use of specific software for calculation of placental volume.[20]

Three-dimensional power Doppler (3DPD) US and 3DPD histogram analysis can help in quantitative and qualitative assessment of the blood flow within and vascularization of the placenta.[2] Several studies suggest using 3DPD US to estimate first-trimester 3D placental volume and vascular flow indices as predictors of adverse pregnancy outcomes, particularly related to pre-eclampsia, gestational hypertension, and small-for-gestational age babies. These studies show statistically significant earlier detection of changes in blood flow dynamics and volume of the placenta when there are such underlying conditions.[19,21]

The role of 3DPD has also been suggested as a complimentary aid to gray scale and Doppler US for the antenatal diagnosis or exclusion of placentation abnormalities, which will be described later on.[22]

MAGNETIC RESONANCE IMAGING

Normal placenta on MRI has a fairly homogeneous structure with relatively low signal intensity on T1-weighted images and relatively high signal intensity on T2-weighted images. Heterogeneities are sometimes observed later in gestation and in complicated pregnancies. These regions of altered signal intensity correspond to foci of infarction, necrosis, and fibrosis.[23]

The T2-weighted images allow differentiation between the hyperintense placenta and the hypointense myometrium (Fig. 5).[7] Ultra-fast spin-echo T2-weighted sequences are particularly useful because the short imaging time circumvents fetal motion and can effectively serve as an ideal adjunct to US. Fat-suppressed T1-weighted images are very useful for detecting blood products.[24]

Diffusion-weighted MRI (DWI) may have value for the detection of placental abnormalities in intrauterine growth restriction (IUGR). Intrauterine growth restriction is associated with reduced exchange surface areas at the peripheral villi, leading to decrease in membrane diffusive conductance. Among the many causes of diffusion restriction in the placenta on MRI, hematoma and infarctions are most important

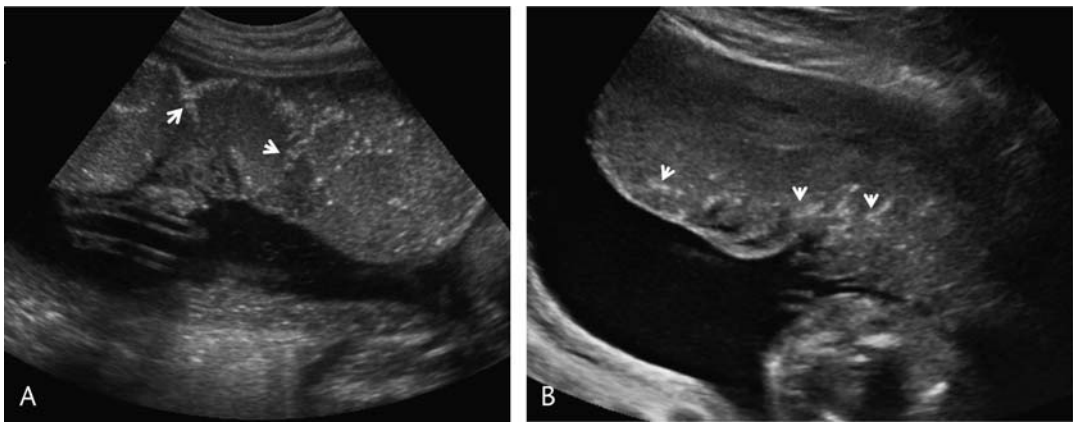

FIGURE 4. Placental calcification. A, Sagittal gray scale ultrasound image of a normal placenta at 35-week gestation shows calcifications (short arrows) in a ''dot-dash configuration'' along the basal plate outlining the cotyledons. B, Transverse gray scale ultrasound image of the placenta at 25-week gestation in a patient with preeclampsia shows premature calcification in a linear pattern suggestive of a distribution in vascular territories (short arrows).

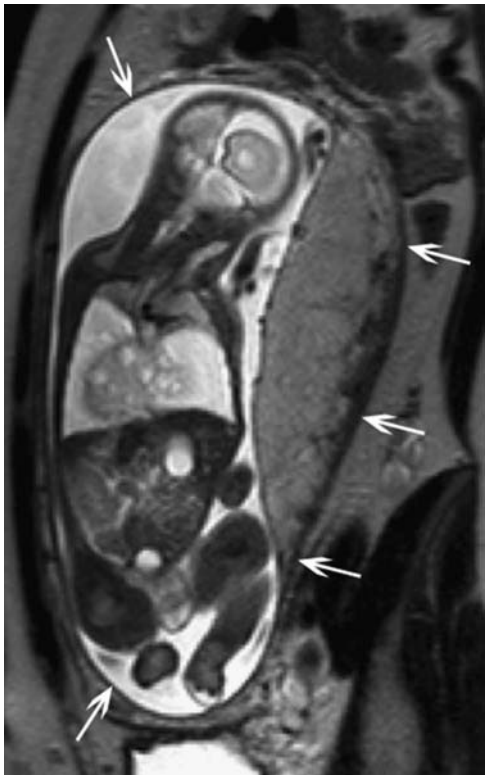

FIGURE 5. Normal magnetic resonance imaging (MRI) appearance of the placenta. T2-weighted sagittal image allows differentiation of the normal hypointense myometrium (arrows) from the relatively hyperintense placenta (P). With placentation abnormalities, this separation of planes is compromised.

because they may lead to dysmaturity of the placenta. This can result in decreased diffusive conductance and restricted blood supply owing to tissue degeneration and scarring. Steady-state free precision (SSFP) imaging can assist in differentiation of abnormal placental vascularity and placental fibrosis.[25,26]

Diffusion-weighted MRI has an emerging role in assessment of placentation abnormalities. On DWI of high b-value, the placenta shows higher signal intensity than the myometrium; therefore, this method can potentially be used for defining the border between the myometrium and the placenta.[27]

Based on ACR recommendations, gadolinium-based magnetic resonance contrast agents should not be routinely administered to pregnant patients. The decision to administer contrast agents to pregnant patients should be based on a risk benefit analysis, with the potential benefits outweighing the theoretical risk of fetal exposure to free gadolinium ions.[28]

COMPUTED TOMOGRAPHY

The first trimester placenta has a relatively homogeneous appearance on CT and is not clearly distinguishable from the myometrium. In the second trimester, the placenta normally becomes increasingly heterogeneous as on other cross-sectional imaging examinations, as maturation continues into the third trimester (Fig. 6).[9]

Computed tomography is not the initial imaging modality of choice in pregnancy because of fetal ionizing radiation exposure. Nevertheless, in emergent circumstances including sepsis and trauma, the clinical benefits of CT can outweigh the risks. For pregnant patients involved in major trauma, in particular, where there is concern for maternal and/or fetal injuries, CT is the mainstay of imaging.[29]

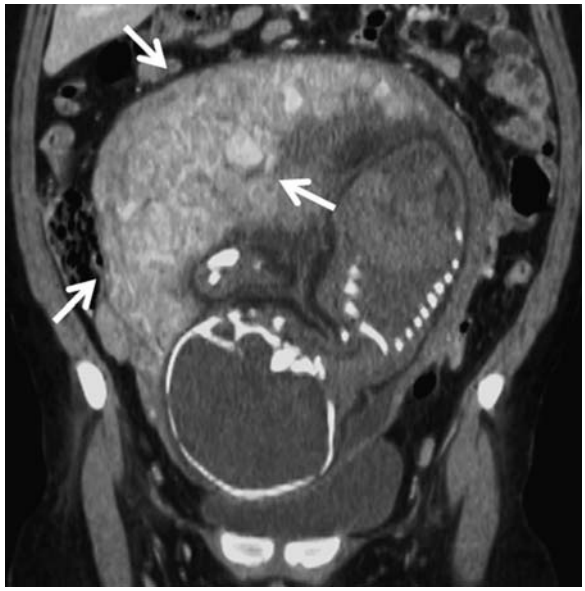

FIGURE 6. Normal computed tomographic appearance of the placenta at 39-week gestation. Coronal contrast-enhanced computed tomographic image obtained after maternal trauma shows expected relatively marked heterogeneous enhancement of the placental tissue (arrows), given the stage of the gestation. There is no evidence of maternal, placental, or fetal injury.

PLACENTAL ABNORMALITIES

Placental abnormalities are related to its morphology, location, extent of placentation, and abruption and very uncommonly due to the presence of tumors (Table 1).

MORPHOLOGICAL ABNORMALITIES

Succenturiate Lobe

The succenturiate placenta is described as one or multiple accessory placental lobes connected to the main placental

TABLE 1. Categories of Placental Abnormalities and Their Associated Pathologies

Abnormalities of the Placenta	
Morphological abnormalities	Succenturiate lobe, circumvallate placenta, placentomegaly, venous lakes, placental infarcts, and intervillous thrombi
Abnormalities of placenta location	Placenta previa
Placentation abnormalities	Placenta accreta, increta, and percreta
Placental abruption	Preplacental, retroplacental, and subchorionic
Tumors	Trophoblastic
	Nontrophoblastic (chorioangioma, teratoma, and metastasis)

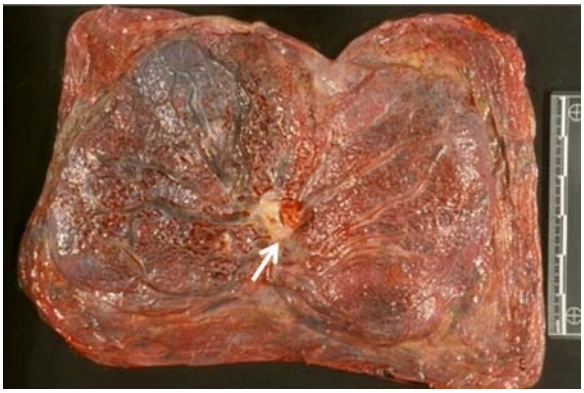

FIGURE 7. Bilobed placenta. Gross pathology specimen of a bilobed placenta at term shows the cord origin (arrow) arising from the space between the 2 lobes.

lobe by blood vessels. Succenturiate lobes occur in approximately 5% to 6% of pregnancies, with an increased incidence with advancing maternal age and in vitro fertilization. A bilobed placenta is a variant where both segments of the placenta are almost equal in size (Fig. 7).[30]

On US, 2 separate placental components are seen with the cord origin usually arising from the main placental lobe (Fig. 8). A succenturiate lobe should be differentiated from a single placenta that extends to 2 quadrants of the uterine cavity. Note should be taken that a myometrial contraction can simulate a succenturiate lobe, and therefore, repeat US can be done to differentiate such a lobe from a focal contraction.[13]

An undetected accessory lobe can remain within the uterus after delivery and can cause postpartum complications. There is an increased association of both vasa previa and velamentous cord insertion with succenturiate placenta.[31]

Circumvallate Placenta

Circumvallate placenta results from insertion of the umbilical cord into the chorioamniotic membrane near the placental edge. Circumvallate placenta has been associated with a higher incidence of placental abruption, premature labor and delivery, IUGR, and perinatal death.[30] A case report suggests that 3D sonographic surface rendering is helpful in the diagnosis of circumvallate placenta.[32]

On US, a circumvallate placenta appears as a 2- to 3-mm irregular, uplifted band of membranes near the placental edge, also described as the "marginal shelf." This band attaches to the placenta within 3 cm of its edge. The marginal shelf can appear as a linear structure protruding into the amniotic fluid and, therefore, can be misinterpreted as a uterine synechia (Fig. 9).[11]

Placentomegaly

On US, the placenta is measured from the subplacental veins, excluding the adjacent myometrium, to the amniotic fluid. A recent study by Lee et al[33] recommends concurrent assessment of placental thickness, placental position, and the fetal gestational age. These authors describe placentomegaly when the anterior placenta is thicker than 3.3 cm and the posterior placenta is thicker than 4 cm.

A thickened placenta is associated with maternal infections, including toxoplasmosis, rubella, cytomegalovirus, and varicella; diabetes and associated fetal macrosomia; Beckwith-Wiedemann syndrome; and fetal hydrops. Placentomegaly is associated with a high risk of placental insufficiency. A thick "jelly-like" placenta, which quivers with abdominal pressure, is associated with a 60% to 75% increased risk of IUGR. A careful fetal growth assessment is required once a thickened placenta is identified on prenatal sonography.[34] A falsely thickened placenta may be seen with a subplacental myoma or with placental abruption when the retroplacental hematoma is isoechoic to the placenta (Fig. 10).

Venous Lake, Intervillous Thrombi, and Placental Infarct

Venous lakes or placental lakes are commonly seen in the third trimester of a normal pregnancy, and are composed

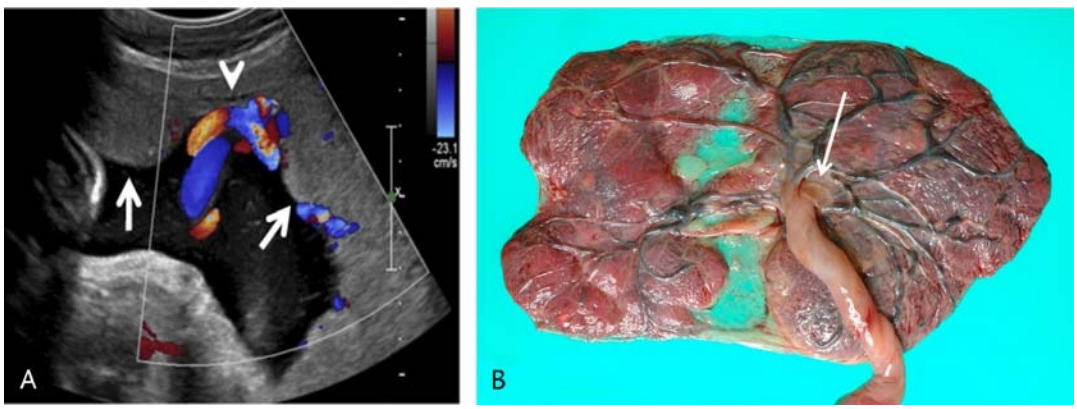

FIGURE 8. Succenturiate placental lobe. A, Transverse color Doppler ultrasound image of a placenta at 26-week gestation shows 2 separate placental lobes (short arrows), with the cord origin (arrowhead) arising from the membranes between them. B, Gross pathology specimen of the placenta of a 34-week gestation fetus with intrauterine fetal demise due to the disruption of the umbilical vessels (kinked by a very large omphalocele, not shown) shows the 2 separate lobes interconnected by a membrane and the cord origin arising from the margin of the dominant placental lobe (arrow).

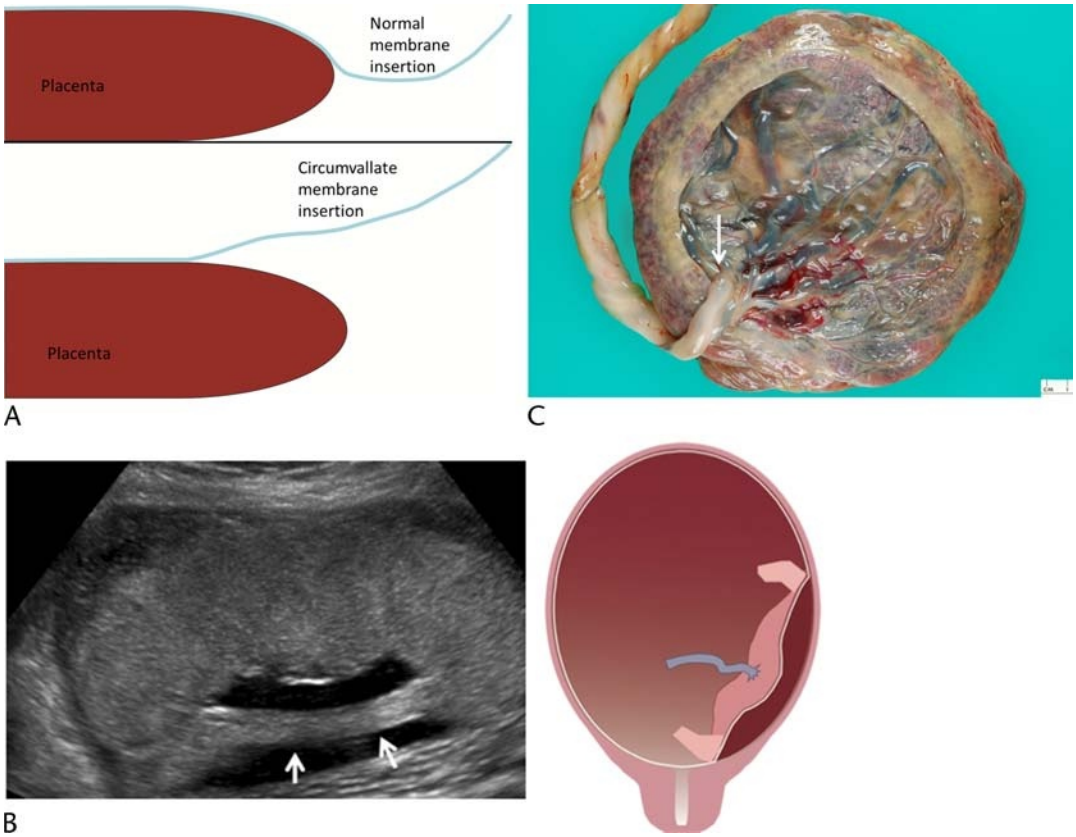

FIGURE 9. Circumvallate placenta. A, Schematic diagram shows normal versus circumvallate insertion of the chorionic membranes. Note that in circumvallate insertion, the uplifted membrane insertion creates a membranous shelf. B, Transverse gray-scale ultrasound image at 30-week gestation shows the band of tissue (short arrows) that creates the ''marginal shelf'' in a circumvallate placenta. Note the ''rolled-up'' edges (marginal shelf, lighter pink color) of the placenta on the corresponding diagram. When imaged in transverse plane, this shelf can appear as a thick continuous band. C, Gross pathology specimen of a 37-week gestation fetus with intrauterine fetal demise due to significant fetal congenital anomalies (not shown) shows abnormal insertion of free membranes near the margin of the placenta. Note the cord inserting into the chorioamniotic membranes, away from the placental tissue or placental edge (arrow).

of small, dilated venules. Venous lakes are clinically significant if seen very early in gestation or if they are larger than 2 cm or greater than 3 in number.[35] On gray scale US, they are hypoechoic and demonstrate internal swirling echoes due to flowing blood. Venous lakes are better seen on higher gain settings and may change size during the same examination.[36] Thrombosed venous lakes, also known as intervillous thrombi, appear hypoechoic on gray scale US. They can be differentiated from venous lakes because they maintain their shape and size on serial sonography at the same sonographic examination. Magnetic resonance imaging is more sensitive for the differentiation between these 2 entities because venous lakes are hypointense on T1-weighted images, whereas intervillous thrombi are hyperintense. Both entities are isointense on T2-weighted images. Additionally, SSFP imaging can assist in distinguishing flowing blood from no flow in a vessel.[37]

Placental infarct refers to a localized area of ischemic villous necrosis. Placental infarctions can occur due to pregnancy-induced hypertension, lupus anticoagulant, low-lying placentas, placental abruption, and viral infections such as with several of the toxoplasmosis, rubella, cytomegalovirus, and varicella infections.[28,29]

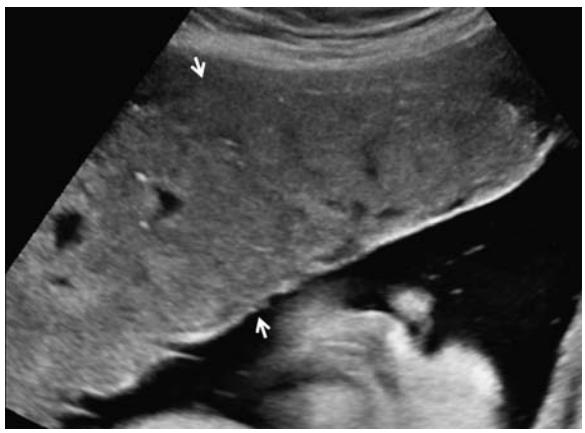

FIGURE 10. Placentomegaly seen at 34-week gestation in a diabetic mother with fetal macrosomia. Transverse gray scale ultrasound image shows a thickened placenta (arrows), [Rev 3, com 9] which measured 6 cm (>4 cm).

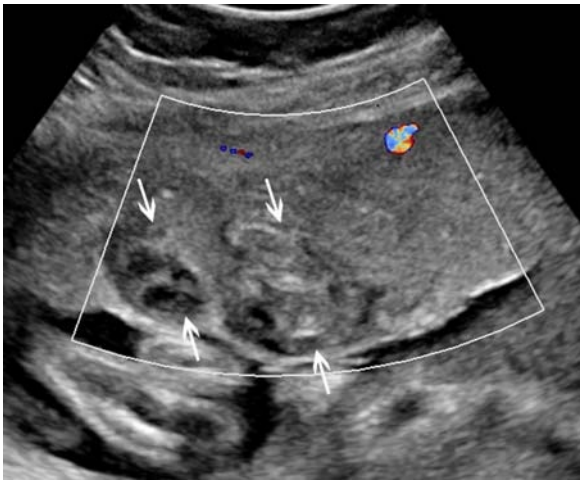

FIGURE 11. Placental infarct. A 35-year-old patient presented at 26-week gestation with severe hypertension and was diagnosed with preeclampsia. Transverse ultrasound image shows placental calcifications and multiple complex cystic lesions throughout the placenta, some of which contain debris (arrows). Doppler US did not show any flow within these cystic lesions. Placental infarction was suspected. Additionally, fetal intrauterine growth restriction was noted on the ultrasound examination. Shortly after the examination, the patient developed fetal decelerations and was delivered emergently. Pathological evaluation of the placenta (not shown) confirmed placental infarctions at various stages.

To our knowledge, the sonographic appearance of placental infarcts has not been well documented. However, they may appear as focal hypoechoic lesions, usually along the placental margins. When infarcts are isoechoic to the placenta, they are more difficult to detect, with a positive predictive value of only around 12%. Infarcts with central bleeding are easier to detect because of the differential echogenicity between the blood clot and the placenta (Fig. 11).[13]

On MRI, placental infarction appears as circumscribed lesions with isointense signal intensity on T1-weighted images and hyperintense signal on T2-weighted and SSFP images. They appear isointense to hyperintense on DWI. Because of the susceptibility effects of blood breakdown products on echo-planar MRI and DWI, these sequences are excellent for the detection of even small hemorrhagic lesions.[38]

Other Rare Morphological Abnormalities

Membranous placenta (placenta membranacea or placenta diffusa) refers to a condition in which all or most of the fetal membranes are covered by chorionic villi because of lack of its differentiation. The thin membranous placenta occupies the entire or most of the periphery of the chorion. It is associated with placenta previa, preterm delivery, IUGR, fetal death, recurrent antepartum hemorrhage, postpartum bleeding, and retained placenta.[39]

Ring-shaped placenta describes an annular-shaped placenta, which may even form a complete ring. However, often, a portion of the placental tissue in the ring is atrophic, resulting in a more commonly seen horseshoe shape. Some believe this entity to be a variant of membranous placenta. This condition is associated with fetal IUGR and antepartum and postpartum bleeding.[40,41]

Fenestrated placenta refers to a condition where the central portion of the discoidal placenta is absent. Often, the defect is villous only, whereas the chorionic plate is intact. However, in rare cases, there may be an actual hole in the placenta. This condition can result in erroneous diagnosis of retained placenta after delivery.[40,41]

Placental mesenchymal dysplasia (PMD) was first described in 1991 and is also known as mesenchymal stem villous hyperplasia. It is a rare placental vascular abnormality characterized by aneurysmal dilation and congestion of chorionic vessels and cystic or hydropic villi.[42] On US, the placenta is enlarged and can contain cystic or "grape-like" component, and therefore, it can resemble partial molar pregnancy on imaging.

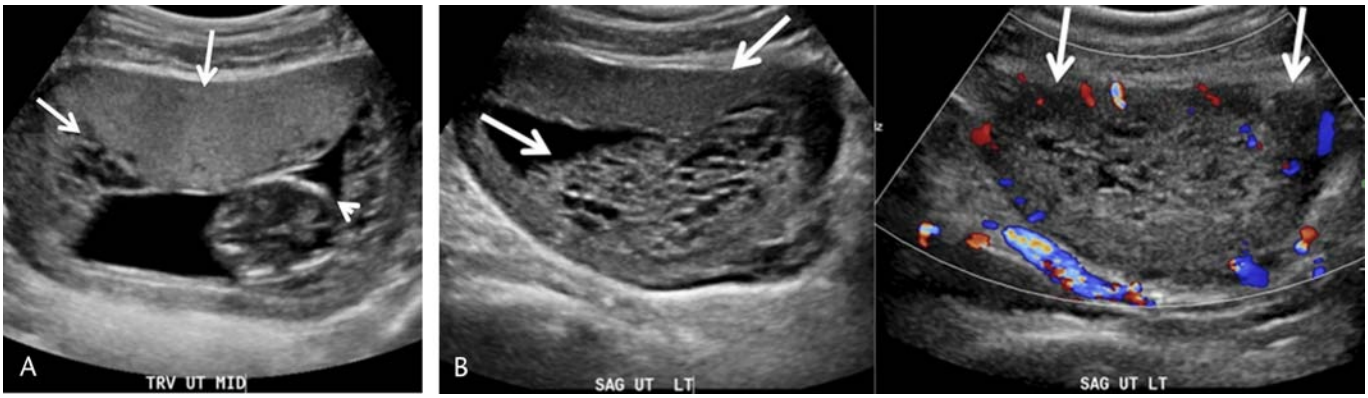

FIGURE 12. Placental mesenchymal dysplasia. A 27-year-old patient presents at 13 weeks gestation for assessment of fetal nuchal translucency. A, Transverse gray-scale image of the placenta showed a cluster of cystic structures associated with the surface and on one side of the placenta (long arrows). A normal-appearing fetus was also identified (arrowhead). B, Sagittal gray scale and color Doppler ultrasound images of this portion of the placenta show a cluster of cystic spaces, which are not of increased vascular flow. Maternal serum alpha-fetoprotein levels were normal. A diagnosis of a partial molar pregnancy along with a normal twin was considered most likely. After an anatomical survey of the viable fetus did not show any structural abnormalities, the pregnancy was managed conservatively with follow-up imaging, After delivery of the fetus, pathological evaluation of the placenta revealed placental mesenchymal dysplasia.

However, unlike a partial mole, a viable fetus can coexist with PMD.[43] One third of PMD cases are associated with Beckwith-Wiedemann syndrome. Additional reported findings in a phenotypically normal fetus include IUGR, preterm delivery, intrauterine fetal demise, and neonatal death. Commonly, the maternal serum alpha-fetoprotein levels are elevated. In 9% of reported cases, associated maternal gestational hypertension, pre-eclampsia, eclampsia, and HELLP syndrome has been observed.[42] Differential diagnosis for PMD includes partial molar pregnancy in a twin gestation with a viable fetus, chorioangioma, and subchorionic or preplacental hemorrhage. The final definitive diagnosis requires pathological evaluation of the placental tissue, often after delivery of the viable fetus (Fig. 12).[43]

ABNORMALITIES OF PLACENTAL LOCATION

Placenta Previa

Placenta previa describes a range of placental locations, when placental tissue either covers or is within 2 cm of the internal cervical os.[44] It is an important cause of third trimester "painless" bleeding, ranging from spotting to severe hemorrhage. Placenta previa complicates 0.3% to 0.5% of pregnancies, with an increased incidence in women with a history of cesarean delivery, a history of termination of pregnancy or uterine surgery, smoking, advanced maternal age, multiparity, cocaine use, and multiple gestations.[45]

Placenta previa was previously graded based on the distance between the placental edge and the internal cervical os. A complete placenta previa is defined as when the internal cervical os is completely covered by placental tissue. In partial placental previa, the internal cervical os is only partially covered by placental tissue. A marginal placenta previa is described as the placental edge reaching up to but not covering the os, while low-lying placenta lies in the lower uterine segment within 2 cm from the cervical os (Fig. 13).[46] The Eunice Kennedy Shriver National Institute of Child Health and Human Development hosted a workshop regarding indications for US and MRI in pregnancy in December 2012, the results of which were published by the American College of Obstetrics and Gynecology. Based on the conclusions of this panel,

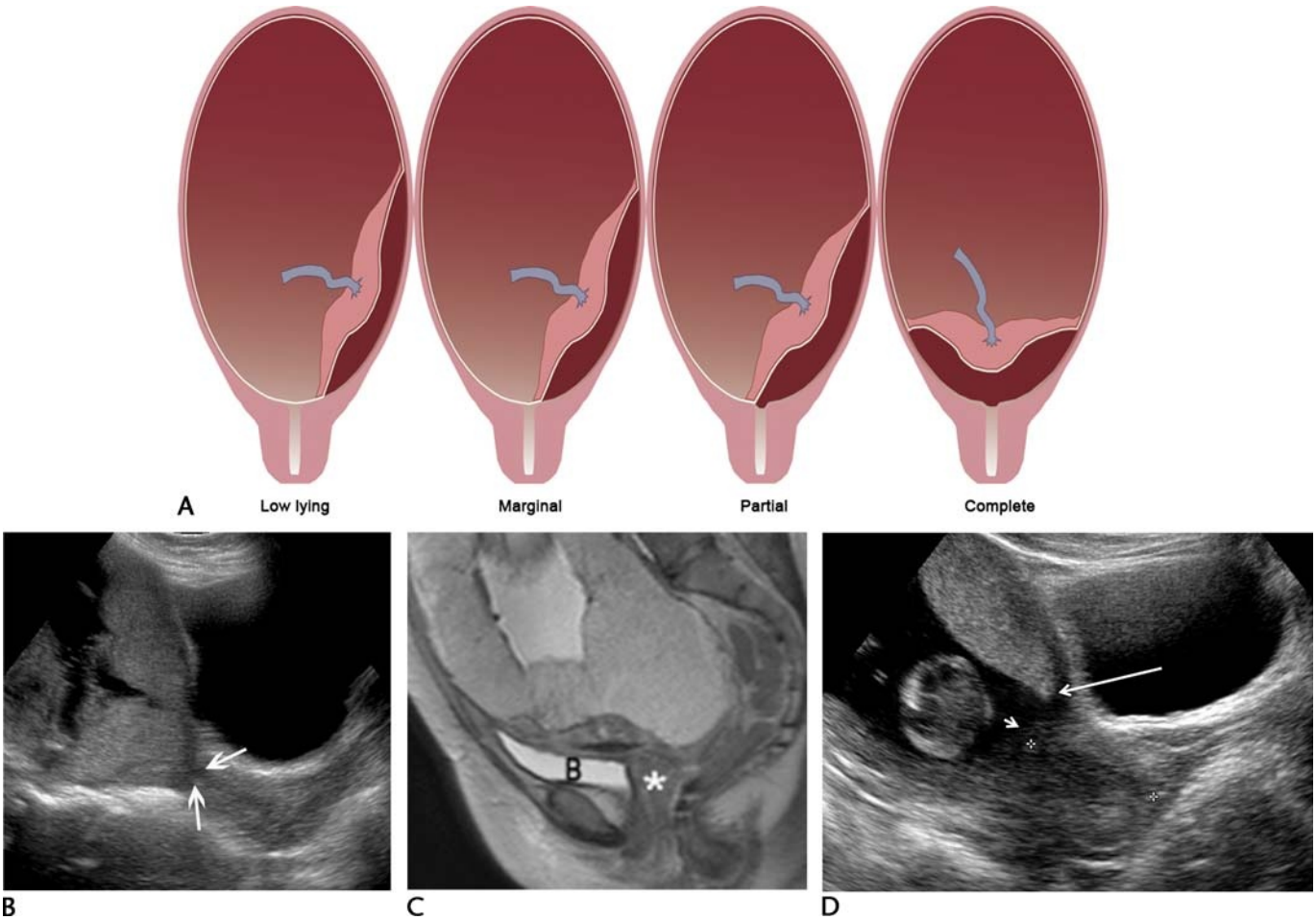

FIGURE 13. Placenta previa. A, Schematic diagram shows variations of placenta previa. B, Sagittal gray-scale ultrasound image at 20-week gestation shows a complete placenta previa covering the internal cervical os (short arrows). C, Sagittal T2-weighted MR image in a different patient at 33-week gestation obtained for assessment of placentation abnormality shows a complete placenta previa (arrows). D, Sagittal gray-scale ultrasound image at 12-week gestation shows a low-lying placenta, with the placental edge (long arrow) within 2 cm of the internal cervical os (short arrow). Calipers denote the length of the cervix.

differentiation between marginal and partial placenta previa is difficult, and the classification of placenta previa was revised. The panel eliminated the terms partial and marginal, while retaining only placenta previa and low-lying placenta. The panel recommended description of the location of the placental edge in relation to the cervix in such cases.[47]

Transabdominal US can result in inaccurate measurements secondary to inadequate visualization of the lower uterine segment due to overlying by the fetal head, maternal obesity, and an overfilled or underfilled maternal bladder.[48] Transvaginal US is the reference standard for assessing the exact location of the placental edge in relation to the internal cervical os.[49] Translabial US can also be used for this purpose if transvaginal US is clinically not feasible.[45]

Vergani et al[50] recently described a classification of placental management based on transvaginal US performed within 28 days of term:

- Placental margin 20 mm away from the internal os; cesarean section is not indicated.
- Placental margin 11 to 20 mm from the internal os; lower likelihood of bleeding, and there may be a need for cesarean section.
- Placental margin 0 to 10 mm from the internal os; higher likelihood of bleeding, and there is a need for cesarean section.
- Placental overlap of the internal os by any distance; cesarean section is definitively indicated.

The placenta migrates greater than 1 mm per week, and therefore, observance of a placenta previa in the first trimester on sonography is not of clinical significance.[45] Placenta previa is usually diagnosed in the second trimester on sonography and requires reassessment in the third trimester to define the final location of the placental margin. Placenta previa is also associated with vasa previa.[51] Screening of patients at risk for vasa previa in the second trimester with transvaginal Doppler, including those with a resolved placenta previa, improves neonatal survival.[52]

PLACENTATION ABNORMALITIES

Placenta Accreta, Increta, and Percreta

Placentation abnormalities are the result of abnormal growth of chorionic villi beyond the decidua basalis, leading to adhesion of the placenta to the uterus, and subsequent failure of placental separation at the time of delivery.[53]

The reported incidence of placentation abnormalities has increased from approximately 0.8 per 1000 deliveries in the 1980s to 3 per 1000 deliveries in the past decade. This is likely due to an increased number of performed cesarean sections and placenta previa[53,54]

Risk factors for placentation abnormalities are divided into major and minor categories. Major risk factors include placenta previa and a history of cesarean section. Minor risk factors include multiparity, uterine surgeries including hysterotomy, dilatation and curettage, and uterine structural anomalies.[55]

Complications associated with placentation abnormalities include potentially life-threatening maternal hemorrhage, disseminated intravascular coagulation, acute respiratory distress syndrome, renal failure, and potentially maternal and fetal death.[46] Because of such detrimental consequences of placentation abnormalities, Baughman et al[56] in their study recommended routine placental US screening in all patients with a history of placenta previa or cesarean delivery.

Placenta accreta constitutes 75% of all placentation abnormalities.[56] It refers to extension of placental tissue to the myometrium without actual invasion (Fig. 14).

More invasive types of placentation include placenta increta and percreta. Placenta increta refers to invasion of placental tissue into the myometrium without extension beyond the serosal surface, whereas placenta percreta refers to invasion of placental tissue beyond the uterine serosa (Fig. 14). In the latter condition, adjacent structures, such as bladder, bowel, and abdominal wall, can be invaded.[7] A mnemonic to remember

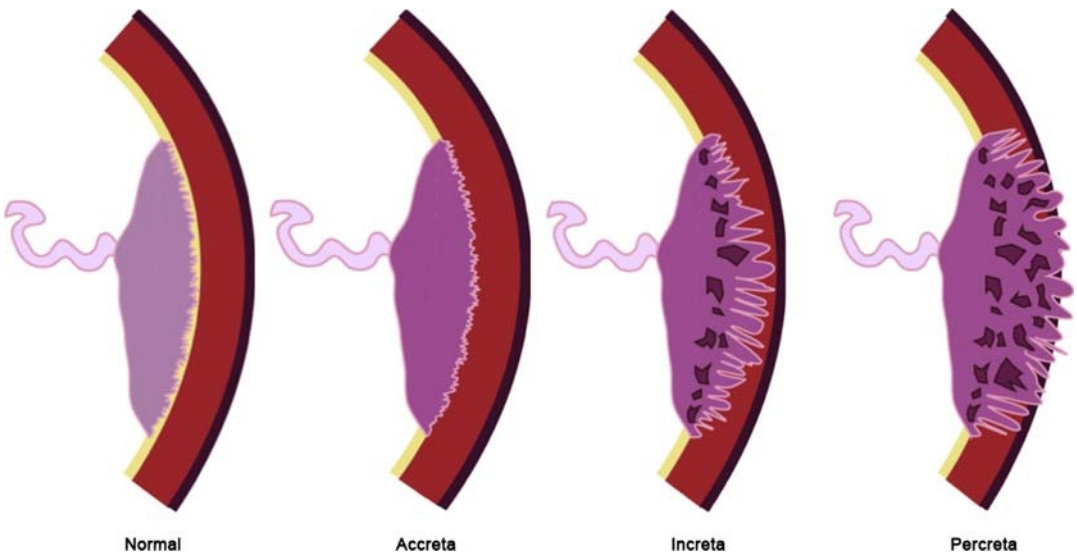

FIGURE 14. Placentation abnormalities. Schematic diagram shows variations of placentation abnormalities resulting from extension of the placental tissue into the myometrium.

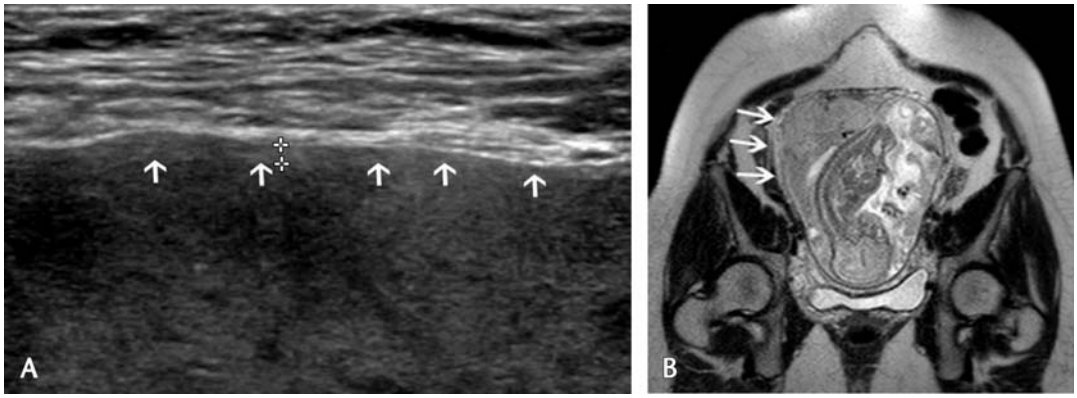

FIGURE 15. Placenta accreta. A, Transverse gray-scale ultrasound image obtained with a linear transducer along the anterior aspect of the uterus in a patient at 32-week gestation with complete placenta previa (not shown), shows progressively deficient myometrium (short arrows) extending to the bladder and uterine interface (not shown here). B, Coronal single-shot fast spin-echo MR image in a patient with constant left-sided abdominal pain during pregnancy, whose ultrasound examination was concerning for placenta accreta, shows gradual thinning with subsequent disappearance of the hypointense signal of the subplacental myometrium off to the right (arrows). Both cases were proven positive for placenta accreta at the time of cesarean delivery.

various classifications includes, accreta (at), increta (in), and percreta (penetrate through).[57]

Ultrasound is the initial imaging modality for the diagnosis of placentation abnormalities. Magnetic resonance imaging is usually reserved for cases with a high clinical suspicion and/or when US findings are negative or ambiguous. However, with a posteriorly implanted placenta, US may not be as effective in visualization of the placental-myometrial interface, and therefore, MRI is considered a more effective diagnostic tool[58] (Fig. 15).

In a recent study, Mclean et al[59] retrospectively evaluated the incremental benefits of MRI after US in a large cohort of gravid patients who were at risk for placenta accreta. These investigators noted that regardless of the MRI findings, the study patients underwent cesarean hysterectomies based on a high clinical suspicion; therefore, they concluded that the role of MRI in assessment of placenta accreta remains uncertain since it does not appear to affect clinical management.

Both gray scale and Doppler sonography play an important role in the diagnosis of placentation abnormalities. On US, the subplacental clear space or hypoechoic zone is well visualized. Implantation of the gestational sac low within the uterine cavity, loss of retroplacental hypoechoic zone, multiple interplacental vascular lacunae, and abnormal interface between the uterus and adjacent organs are the best US markers for placentation abnormalities.[57] Comstock et al[60] reported a diagnostic sensitivity of 7% and a positive predictive value of 14% with gray scale US for the detection of an absent subplacental hypoechoic zone. However, with the addition of Doppler, the finding of linear vascular channels extending from the placental parenchyma into the myometrium increased the sensitivity and positive predictive value to 50% and 88%, respectively. These vascular channels can be distinguished from normal vascular lakes by their bizarre, irregular shapes and by more turbulent flow. Their appearance is in sharp contrast to vascular lakes, which are smooth and well defined, with laminar flow on Doppler.[61] Comstock et al[60] also suggested that the finding of linear vascular channels is the most predictive sonographic sign of placenta accreta, with a sensitivity of 79% in the second trimester and with a sensitivity of 93% in the third trimester.

Recent studies describe the complimentary role of 3D power Doppler (3DPD), which more effectively depicts the vascular changes associated with this condition.[62,63] Shih et al[64] described the following 3DPD criteria in placentation abnormalities: intraplacental hypervascularity, inseparable cotyledonal and intervillous circulations, and tortuous vascularity with irregular branching and multiple coherent vessels seen on the basal view, with the latter being the best single criterion for the diagnosis of placenta accreta, with a sensitivity of 97% and a specificity of 92%.

On MRI, the abnormal placenta appears heterogenous on T2-weighted images and contains dark intraplacental bands, which extend into the myometrium. Their irregularity and random distribution help differentiate them from normal placental septa. The uterus can bulge and lose its normal gravid appearance, which has been likened to an "inverted pear shape." Focal regions of myometrial interruption are best seen on T2-weighted images. Direct invasion into the surrounding organs is also best depicted on T2-weighted images. If the bladder is invaded, "tenting" of the bladder wall may be observed (Fig. 16).[56–58]

PLACENTAL ABRUPTION

Placental abruption is defined as premature separation of the placenta from the uterine wall. It is a well-known cause of vaginal bleeding in the latter half of the pregnancy, with a prevalence of less than 1% of gestations.[65]

Maternal conditions associated with abruption include hypertension, pre-eclampsia, abdominal trauma—especially for anterior placentas, cocaine abuse, cigarette smoking, alcohol use, and advanced maternal age.[66]

Abruption can occur at various placental locations including preplacental, subchorionic, and retroplacental. Preplacental abruptions are less common than the other types and

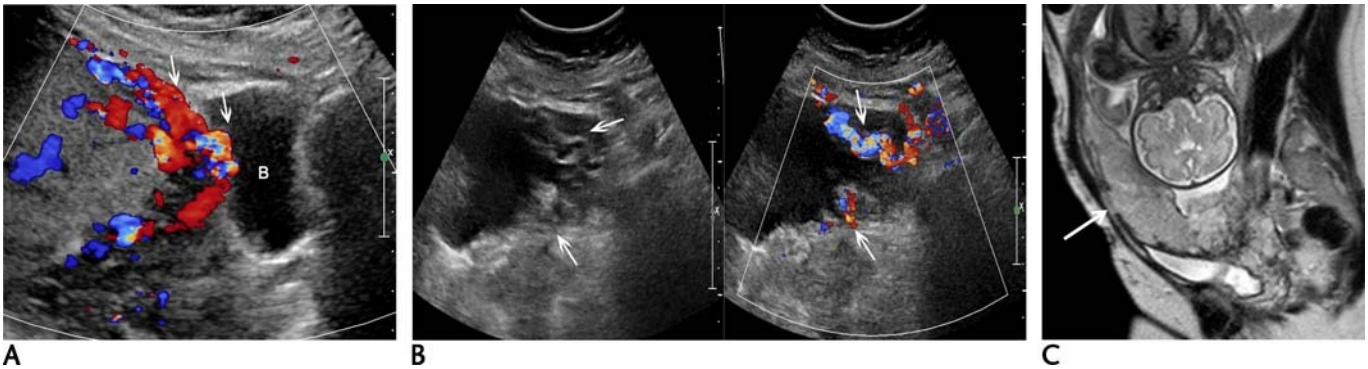

FIGURE 16. Placenta percreta associated with complete placenta previa in a patient with history of 2 prior cesarean sections. A, Gray-scale sagittal Doppler ultrasound image shows a gradually thinning myometrial layer (arrows), which becomes indistinguishable between the uterus and the bladder (B) interface, with an absent subplacental clear space (PL: placenta). B, Sagittal gray-scale and color Doppler images in the same patient show numerous irregular vessels with turbulent flow extending into the bladder wall (arrows). At delivery, the patient underwent cesarean section and hysterectomy, as well as bladder repair. C, Sagittal single-shot fast spin-echo MR image in a different patient at 28 weeks gestation, with previous cesarean sections shows invasion of the placenta into the anterior abdominal wall just above the cesarean section scar and focal interruption of the myometrium (arrow). Placenta percreta was confirmed at surgery and on pathologic evaluation.

occur under the chorionic plate between the placenta and amniotic fluid.[67]

Retroplacental hematomas occur secondary to rupture of decidual arterioles, which result in accumulation of blood between the basal plate of the placenta and the uterine wall. Consequently, the placenta is at risk of basal plate necrosis and villous infarction, with serious adverse pregnancy outcomes.[25] The clinical significance of such hemorrhage is related to the gestational age at the onset and the size of the hematoma. Smaller hematomas have a larger impact in early gestation, when the fetus is less than 20 weeks old. In later stages of gestation, hematomas are of significance if they are large enough to strip more than 50% of the placenta from the myometrium.[68]

Subchorionic hematomas result from abruption of the placental margin. This location is preferred for accumulation of hemorrhage since the placenta is firmly attached to the myometrium, whereas the membranes are easily separable.[69]

The retroplacental complex normally measures 1 to 2 mm in thickness and is composed of uteroplacental vessels and myometrium. Retroplacental hemorrhage should be suspected if this region is thickened on US.[11] Acute hemorrhage is hyperechoic to isoechoic to the placental tissue, becomes hypoechoic a week after it has occurred while resolving hematoma, and appears sonolucent after the second week (Fig. 17).[69]

Ultrasound is not sensitive for detection of acute or hyperacute placental abruption, especially if the hemorrhage is concealed, that is, if it is confined in the retroplacental location by intact membranes. When a retroplacental hematoma is identified, more aggressive management is required because it may suggest a worse neonatal outcome. Magnetic resonance imaging is highly accurate for the diagnosis of placental abruption and should be considered when there is high clinical suspicion and a negative US examination. Potential advantages of MRI include a higher soft-tissue contrast and the ability to depict blood products, which help differentiate blood from other fluid collections. A study by Masselli et al[70] reported the particular importance of diffusion-weighted MRI for the detection of placental abruption, particularly in sonographically occult retroplacental hematomas. According to this study, DWI is an excellent sequence for detecting intrauterine hemorrhagic lesions due to blood breakdown products causing susceptibility effects. With the ability of MRI to estimate the age of the hemorrhage, it can also be useful in the assessment of a pregnant patient with vaginal bleeding and for identifying the cause of rapidly progressive hematoma, which can result in fetomaternal decompensation.[70]

Computed tomography is mainly used in trauma because of its ability to depict both the pregnancy and the maternal anatomy. The CT appearance of placental abruption is variable. Abruption can appear as an area of absent placental enhancement due to devascularization or as an area of high attenuation related to hemorrhage deep to the placenta or within the amniotic fluid (Fig. 18).[71]

Kopelman et al,[72] in a recent study, described CT as an important tool for accurately identifying placental abruption in late second and third trimester trauma patients and reported that CT helped to stratify the risk for fetal complications. According to their study, the likelihood of the need for delivery after trauma increased when, in particular, 75% or greater of the placenta demonstrated poor enhancement with IV contrast.

PLACENTAL TUMORS

Placental Cyst

Placental cysts may occur under the fetal plate (subchorionic) or within placental tissue (septal). The etiology of these cysts is unknown to our knowledge. These cysts are usually benign and do not alter obstetric management; however, in rare instances, subchorionic cysts, when attached near the umbilical cord insertion, may cause umbilical cord constriction, leading to IUGR.[73]

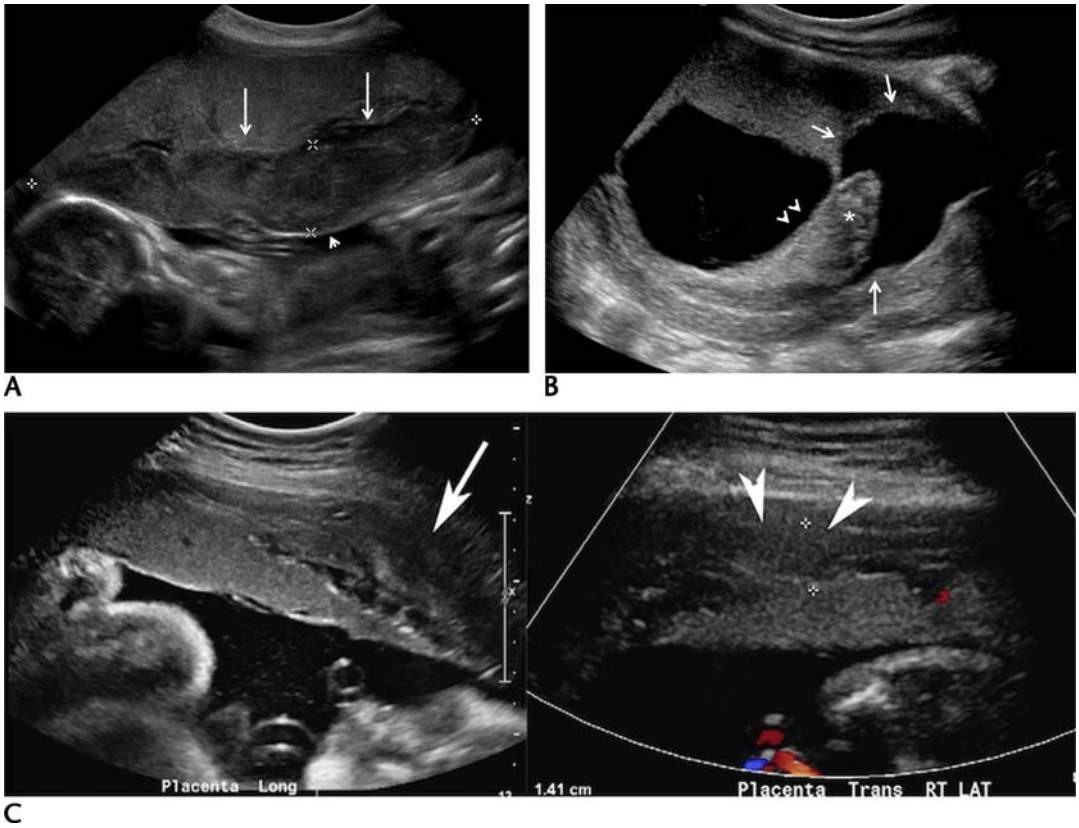

FIGURE 17. Placental abruption. A, Transverse gray-scale ultrasound image at 24-week gestation shows a large hypoechoic hemorrhage between the surface of the placenta (long arrows) and the membranes (short arrow), representing a pre-placental location. B, Sagittal gray-scale ultrasound image at 11-week gestation shows a chronic anechoic hemorrhage in a retroplacental location (arrows), which also extends into the subchorionic space (arrowheads denote the chorionic membranes over the placenta). Note the uplifted placental edge (asterisk) due to the retroplacental hemorrhage. C, [Rev 3, com 13] Sagittal and transverse gray-scale ultrasound images of the placenta in a different patient at 27-weeks gestation who presented with painless vaginal bleeding, shows subacute echogenic hemorrhage (long arrow and arrowheads) in a retroplacental location.

These appear as anechoic thin-walled structures along the placental surface without vascular flow on Doppler imaging, which are readily detectable on US (Fig. 19).

Trophoblastic Placental Tumor

Trophoblastic tumors consist of a heterogeneous group of interrelated lesions arising from the placental trophoblastic tissue. This group includes partial and complete hydatidiform mole, invasive mole, choriocarcinoma, and placental site trophoblastic tumor.[74]

The main risk factors for developing gestational trophoblastic disease are extremes of maternal age and a history of gestational trophoblastic disease. These conditions are usually diagnosed when a greater than appropriate rise in quantitative analysis of urine and serum HCG levels is detected.

Hydatidiform mole is a benign process and can be partial or complete. The presence of fetal parts differentiates partial mole from the complete mole, as no fetal parts are detected in the latter form. Complete hydatidiform moles have a 46XX or 46XY diploid chromosomal pattern (46YY are not viable).[75] All of the chromosomes are paternal in origin and result from fertilization of an egg, which has lost its chromosomes. This egg can be fertilized with 2 sperms, or a single sperm, which then duplicates resulting in 2 sets of chromosomes. The karyotype of a partial mole is a usually triploid (69XXY, 69XXX, or 69XYY) pattern and is the result of fertilization of a normal egg by 2 sperms. There is an increased risk of choriocarcinoma with complete molar pregnancy.[75]

On US, both complete and partial moles are associated with an enlarged uterus. With partial molar pregnancy, an enlarged placenta is detected with multiple diffuse anechoic lesions. The fetus is nonviable with various congenital malformations of the fetal tissue, features of triploidy, and growth restriction (Fig. 20).[76]

Complete molar pregnancy in the first trimester exhibits an enlarged uterus with the endometrial canal filled with heterogeneous vascular hyperechoic tissue. Small cysts may be present at this early phase, which may potentially be confused with an anembryonic pregnancy. Later in the pregnancy, the endometrium is filled with an echogenic mass containing multiple anechoic (cystic) spaces, which are the hydropic villi. This is described as the "Swiss cheese" or "snowstorm" appearance.[76] The ovaries may contain bilateral theca lutein cysts (large cysts with multiple septations), which arise in reaction to the high HCG levels.[76] Doppler evaluation reveals high velocity, low impedance flow in the intrauterine mass, which is contrary

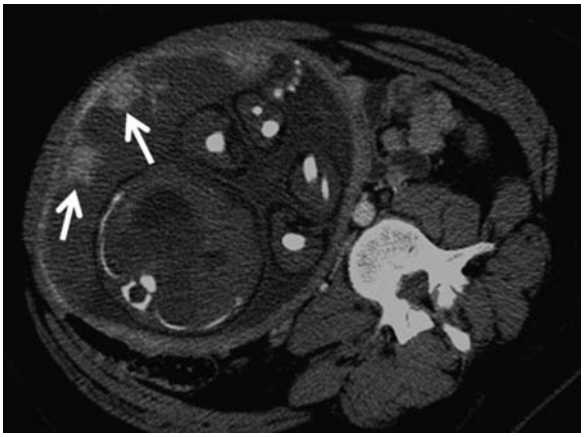

FIGURE 18. Computed tomographic appearance of a placental abruption. A 32-year-old woman at 30-week gestational age, who was involved in high velocity motor vehicle collision, underwent contrast-enhanced computed tomography for trauma evaluation. Axial computed tomographic image obtained through the abdomen shows a largely devascularized placenta with few remaining areas of enhancement (arrows), representing substantial posttraumatic placental abruption. The patient also had traumatic brain injury, multiple facial fractures, pelvic and rib fractures, and a liver laceration (not shown). An emergent cesarean section was performed to rescue the fetus.

to normal uterine arcuate arterial flow of low velocity and high impedance.[77] Coexistent mole and pregnancy may be seen in a dizygotic twin pregnancy, in which case, the normal fetus has normal placenta, differentiating it from a partial mole.[78]

To our knowledge, MRI has no established role in the initial diagnosis of hydatidiform moles.[54] It is useful in malignant forms of gestational trophoblastic neoplasia for characterization of the degree of myometrial and/or parametrial invasion, as in the assessment of treatment response. On MRI, an isointense intrauterine mass is seen on T1-weighted images, which may contain hyperintense areas representing hemorrhage. The intrauterine mass is markedly hyperintense on T2-weighted images and demonstrates avid contrast enhancement with numerous small cystic regions (Fig. 21).[58]

Computed tomography has limited use in imaging of less aggressive molar pregnancy. Contrast-enhanced CT shows an enlarged uterus with an endometrial mass, with focal irregular low attenuation areas and a reticular pattern of enhancement.[76]

Hydatidiform moles can potentially differentiate into an invasive mole, placental site trophoblastic disease, or choriocarcinoma.[77]

Invasive mole and choriocarcinoma are manifestations of persistent trophoblastic proliferation. Continuous rise in HCG levels after treatment of molar pregnancy, an ectopic pregnancy, or an abortion suggests a persistent trophoblastic process.[75]

An invasive mole is distinguished by excessive trophoblastic overgrowth and extensive penetration of trophoblastic elements, including whole villi deep into the myometrium. Unlike choriocarcinoma, this persistent trophoblastic process is locally invasive and rarely metastasizes.[14]

Ultrasound is the initial examination of choice for assessment of invasive molar pregnancy. Doppler imaging demonstrates an invasive mass with high-velocity, low-impedance flow, which has a lower resistive index compared with a complete mole.[78]

Magnetic resonance imaging is excellent in demonstrating the depth of myometrial invasion because of its superior soft-tissue resolution. On T2-weighted imaging, tumors typically have heterogeneous signal intensity, with an indistinct boundary between the endometrium and myometrium. Diffusely increased myometrial signal with obliteration of the normal zonal architecture on T2-weighted imaging may reflect diffuse myometrial involvement by tumor, although it has also been shown to occur with missed and incomplete miscarriage and in patients who have had a recent diagnostic curettage (Fig. 22).[76,77]

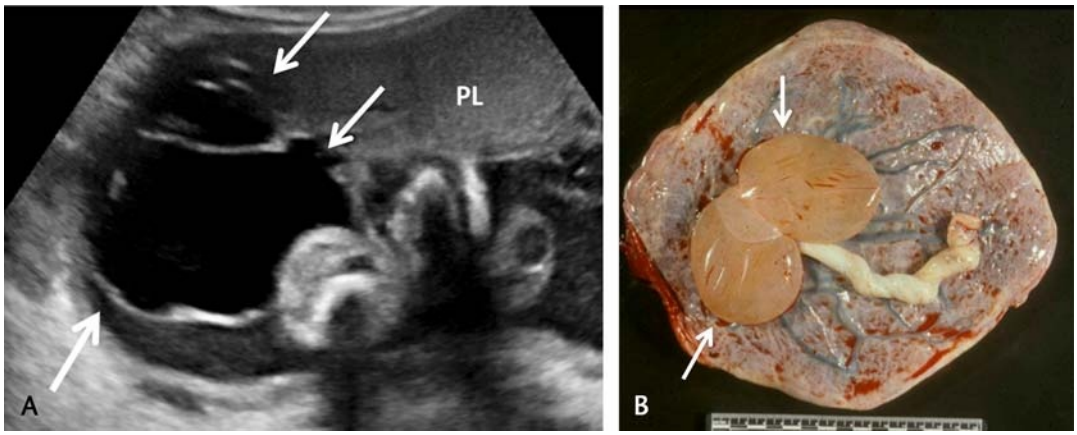

FIGURE 19. Placental cyst. A, Gray scale transverse ultrasound image at 24-week gestation shows a thin-walled anechoic subchorionic cyst (arrows) arising from the chorionic surface of the placenta (PL). This patient had an uneventful pregnancy. B, Pathological specimen of a 35-week gestation fetus with intrauterine fetal demise secondary to cord thrombosis (not shown), shows a lobulated placental surface subchorionic cyst near the cord origin (arrows).

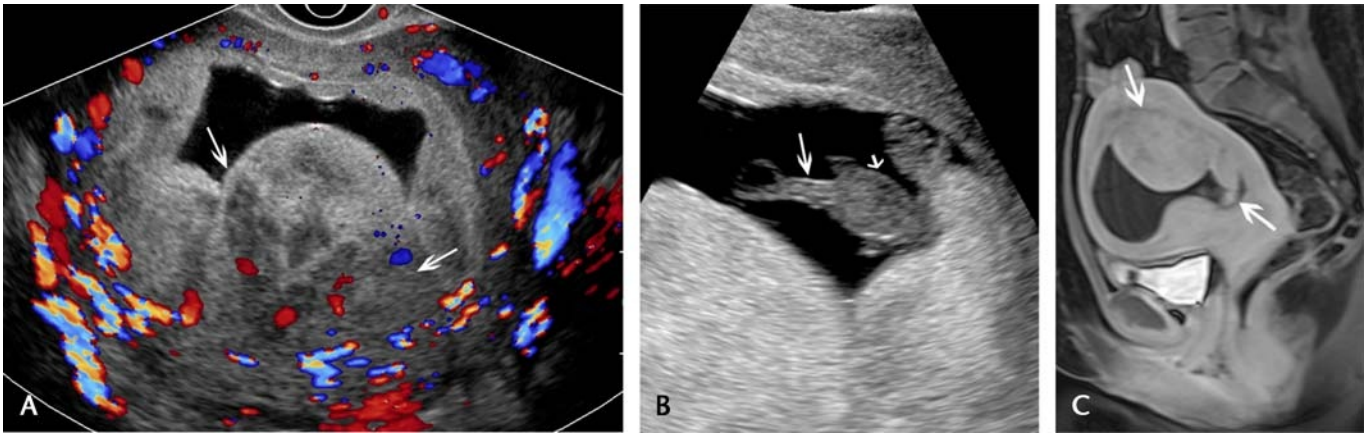

FIGURE 20. Partial hydatidiform mole. A 29-year-old women with unknown last menstrual period was referred for assessment of fetal gestational age. A, Transverse Doppler ultrasound image of the uterus shows a heterogeneous solid and cystic vascular mass within the endometrial cavity (arrows). The uterine cavity also contains fluid. B, In addition to the vascular mass, fetal parts (small arrow), and a short umbilical cord (long arrow) were also identified. C, [Rev 3, com 15] On MRI, sagittal fat-suppressed T2-weighted image of the pelvis shows the heterogeneous mass within the uterine cavity (top arrow) and fetal parts (bottom arrow).

Computed tomography is not routinely done for evaluation of invasive mole. However, a study by Miyasaka et al[79] described the CT findings of areas of myometrial enhancement, with filling defects, as indicative findings for the diagnosis of invasive mole on CT.

Choriocarcinoma occurs rarely and has a variable appearance on US. If detectable, it can present as a small tumor or as a large intrauterine heterogeneous infiltrating mass containing cystic areas and hemorrhage. Choriocarcinoma may be difficult to differentiate from an invasive mole, with presence of metastases as the only distinguishing feature.[77]

Magnetic resonance imaging has an adjunctive role after US for the evaluation of local invasion of the primary intrauterine tumor.[80] Choriocarcinoma is usually seen as an intrauterine mass with heterogeneous high signal intensity on T2-weighted images and marked enhancement on postcontrast images, findings that reflect the high vascularity of the tumor. Tumor vascularity can also be reflected by focal signal voids on T1- and T2-weighted magnetic resonance images. Myometrial invasion is visible as high–signal intensity foci within the myometrium, which demonstrate enhancement on postcontrast images. Enhancing parametrial soft tissue is characteristic of local spread.[80] Magnetic resonance imaging can also help depict metastatic disease, particularly within the pelvic organs and lymph nodes.[14]

Computed tomography is reserved for detecting distant metastases and, therefore, is important in the staging process.[14] Choriocarcinoma is typically seen as a nonspecific enhancing heterogeneous soft-tissue density in the uterus. Common sites of metastases include lungs, brain, liver, and GI tract. Lung metastases are the result of embolized trophoblastic tissue through the venous system draining the uterus. These lesions are usually small and may cavitate centrally. Because these lesions are vascular, they may be surrounded by

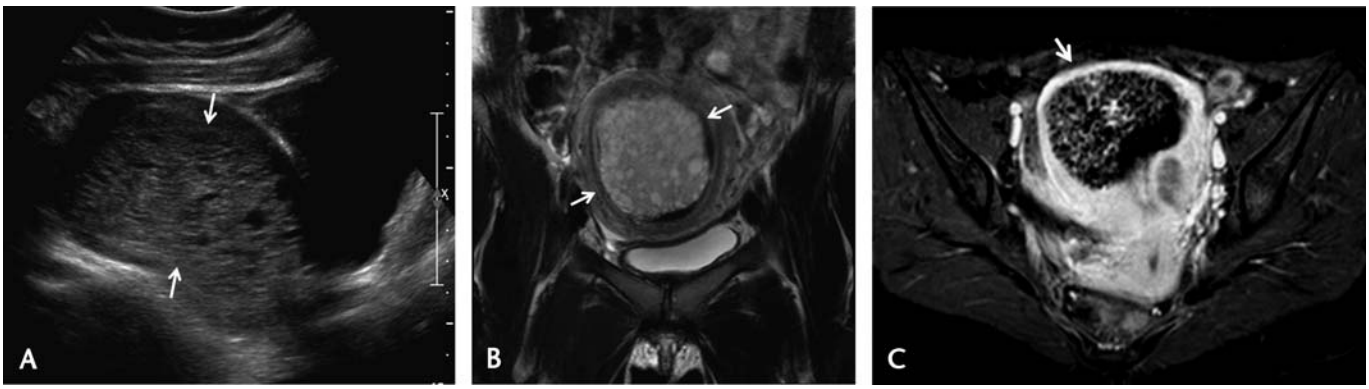

FIGURE 21. Complete hydatidiform mole. A, Sagittal gray scale ultrasound image at 10-week gestation in a patient with a markedly elevated human chorionic gonadotropin (HCG) level shows a cystic mass filling the entire endometrial cavity (arrows). During the ultrasound examination, there was suspicion for myometrial invasion (not shown), and an MRI was requested for clarification. B, Coronal T2-weighted magnetic resonance image shows the heterogeneous cystic mass filling the endometrial cavity (arrows). C, T1-weighted axial postcontrast administration magnetic resonance image in the same patient shows enhancement of the mass with apparent cystic spaces. There was no evidence of myometrial invasion on MRI. The diagnosis of a complete molar pregnancy was confirmed on pathological evaluation (not shown).

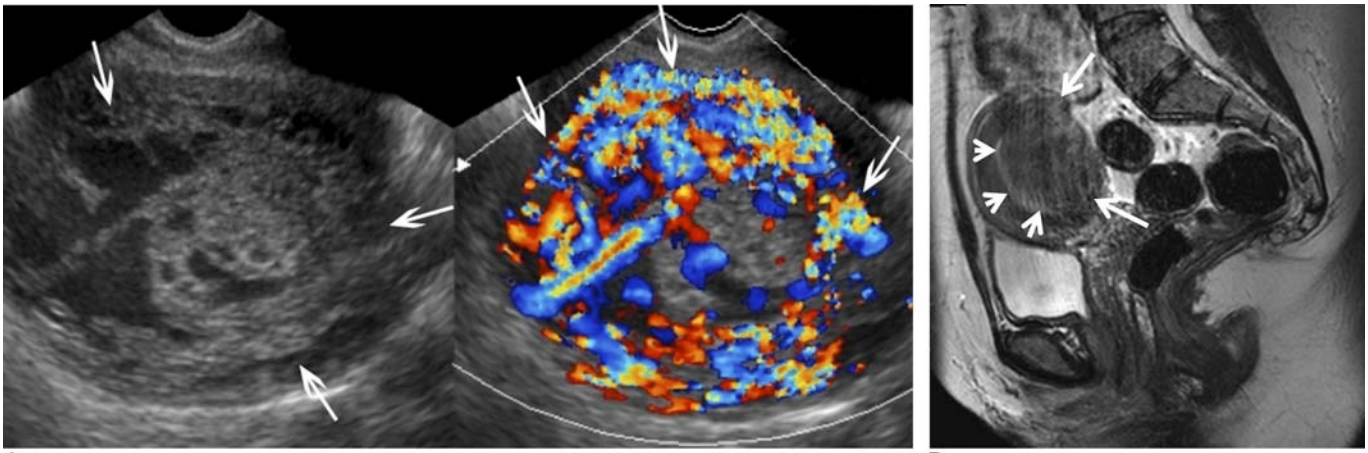

FIGURE 22. Invasive molar pregnancy. A 35-year-old woman with a history of complete molar pregnancy, who was treated with dilatation and curettage, now presents with persistently elevated HCG levels. A, Gray scale and color Doppler transverse ultrasound images of the uterus at the same level show a distended endometrial cavity filled with heterogeneous vascular soft tissue and loss of distinction between endometrium and myometrium (arrows). Massively enlarged multicystic ovaries (theca-lutein cysts) were also seen on the ultrasound examination (not shown). B, Sagittal T2-weighted magnetic resonance image in a different patient shows a low signal intensity mass projecting into the endometrial canal (short arrows) with loss of the posterior junctional zone (long arrows). The mass proved to be an invasive mole.

hemorrhage, resulting in ground glass appearance.[75] Liver metastasis are indicative of a poor prognosis because they appear later in the course of the disease. The liver lesions are vascular with avid enhancement with contrast material. These lesions may also have a hemorrhagic component or bleed.[81]

A few rare cases of intraplacental choriocarcinoma have been reported in live term pregnancies. These case reports emphasize the need for complete pathological assessment of the placenta of normal term pregnancies with high clinical suspicion and any gross placental anomaly,[82] as well as the need for a thorough search for metastases in the mother and fetus in the setting of intraplacental choriocarcinoma.[83]

Placental site trophoblastic tumor is histopathologically composed of intermediate trophoblastic cells. It can be seen in association with gestational trophoblastic disease and after a normal pregnancy or a miscarriage. It usually presents with vaginal bleeding.[78] On US, this tumor can appear as a hyperechoic heterogeneous mass with cystic spaces similar to other trophoblastic pathology. They can have hypervascular and hypovascular components. Serial assessment of uterine volume, as well as assessment with color Doppler US, are important in monitoring response to treatment.[81]

Nontrophoblastic Placental Tumor

Placental chorioangioma is a rare benign vascular mass with fetal blood supply. It is most commonly located on the fetal side of the placenta near the cord insertion. Clinical significance is size dependent. Lesions greater than 5 cm can lead to polyhydramnios or oligohydramnios, hemorrhage, premature delivery, premature placental separation, and placenta previa. These manifestations may result in severe fetal distress and intrauterine death. Chorioangioma may also lead to nonimmune hydrops fetalis. The size and vascularity of the chorioangioma increase with advancing gestational age. Therefore, once detected, close follow-up of the pregnancy is warranted.[84]

Ultrasound typically depicts a well-defined hypoechoic mass, with or without calcifications, bulging on the fetal

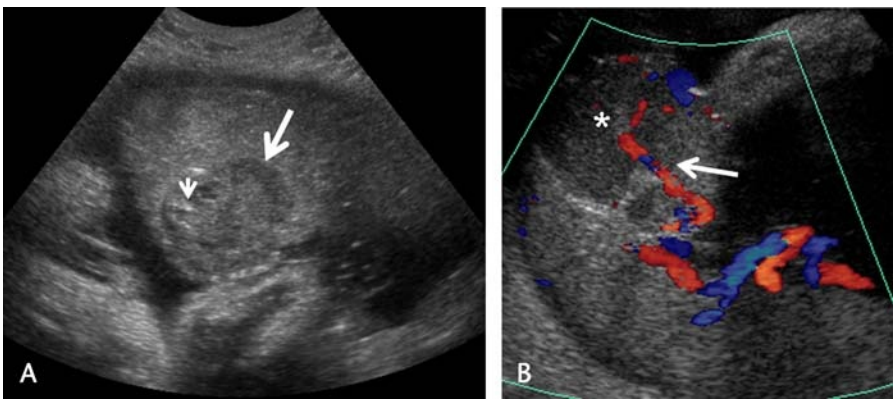

FIGURE 23. Chorioangioma. A, Gray-scale transverse ultrasound image at 35-week gestation shows a hypoechoic mass near the chorionic plate of the placenta (long arrow). Note the tiny calcifications in the mass (short arrow), which are a relatively rare finding in chorioangioma. B, Sagittal color Doppler ultrasound image in the same patient shows vascularity of the mass (asterisk), with a dominant feeding vessel branching into the lesion (arrow). The pregnancy progressed normally, and the fetus was born at full term. Chorioangioma was confirmed on histological analysis of the placenta (not shown).

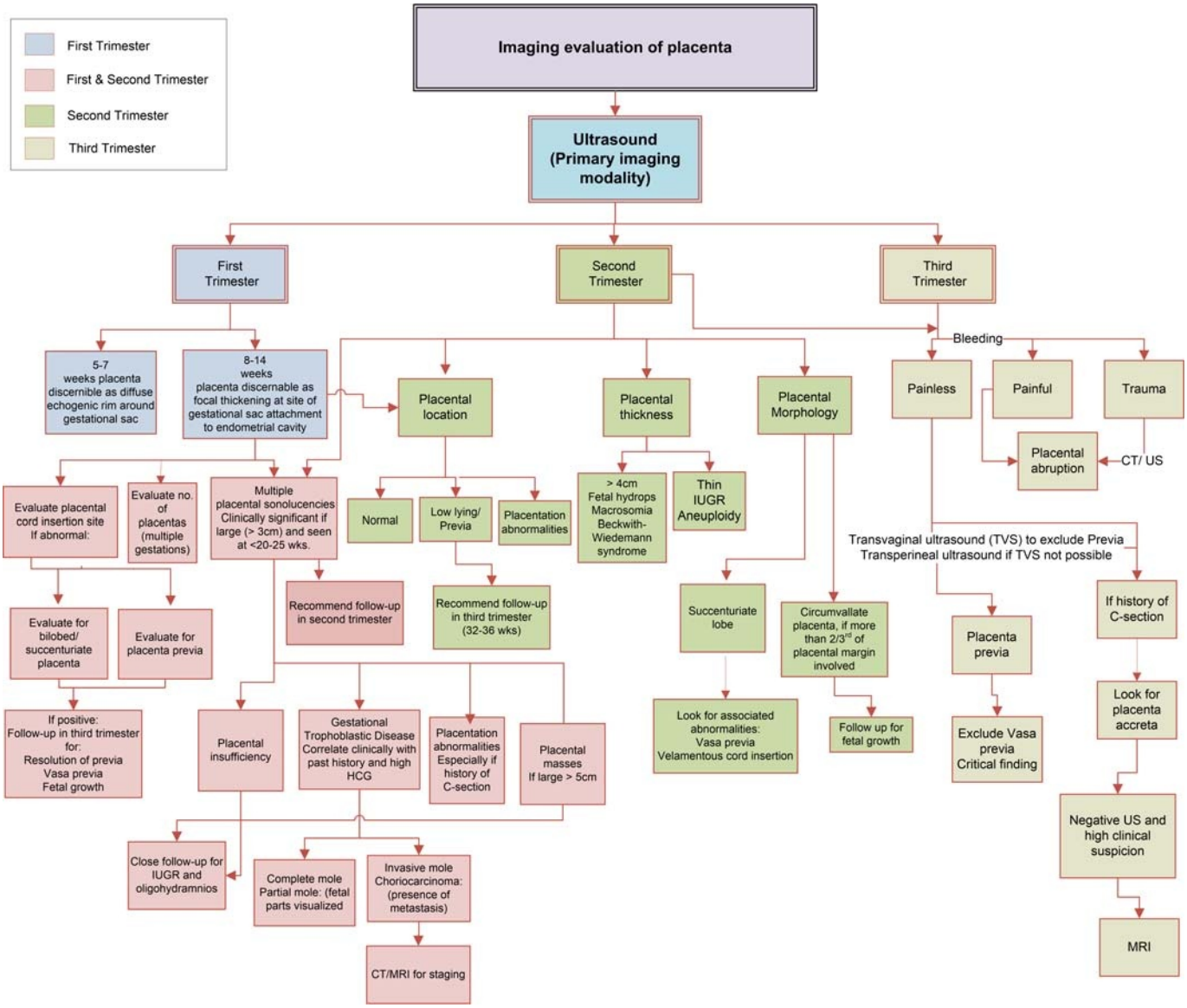

FIGURE 24. [Rev 3, com 16] Suggested algorithmic approach to imaging evaluation and management of placental abnormalities based on our experience. The algorithm provides a generalized road map for approach to placental abnormalities.

surface of the placenta. The presence of calcifications may represent reduced blood flow and may be associated with a better prognosis.[84] Doppler examination demonstrates a variable amount of flow. A large feeding vessel branching into the lesion may be visible (Fig. 23). Detection of high arterial flow is associated with an increased risk of fetal hydrops.[14]

On MRI, T2-weighted images depict a high signal intensity mass with intralesional areas of low and high signal corresponding to areas of hemorrhage.[22]

Placental teratoma is a benign rare placental mass composed of all 3 germ cell layers. It has no clear clinical significance and is not associated with adverse effects on the pregnancy.[85]

On US, placental teratoma appears as a soft-tissue placental mass with variable echogenicity because of the presence of different types of tissue. The presence of calcifications helps differentiate placental teratoma from hydatidiform mole and choriocarcinoma, as the latter 2 masses rarely calcify. Differentiation between an acardiac fetus and a teratoma can be difficult on US because an acardiac fetus may present as a calcified mass. The former may demonstrate fetal poles and an umbilical cord insertion, aiding in differentiation.[85]

Placental metastases are very rare, with most reported cases from melanoma. Other possible sources include maternal sarcoma, breast cancer, ovarian tumors, leukemia, and lymphoma.[86]

Imaging manifestations of metastases to the placenta are nonspecific. On sonography, they may appear as hyperechoic or hypoechoic nodular placental masses. Multiplicity favors this diagnosis; however, such an appearance strongly requires correlation with patient history of a primary malignancy.[14]

CONCLUSIONS

A normal placenta is the link to fetal and maternal well-being and adequate fetal development. Detailed and accurate sonographic assessment of the placenta is mandatory during routine prenatal sonography. Radiologists should be aware of the various types of placental abnormalities and the appropriate further imaging recommendations. To accomplish this, we have provided an algorithmic approach based on our literature review (Fig. 24). The active role of a radiologist in managing placental abnormalities is the key to complete prenatal care.

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