Ultrasound of the Placenta

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Abstract: This review covers ultrasound evaluation of the normal and abnormal placenta with clinical correlation. Normal placental function is essential for a healthy pregnancy outcome as well as for maternal, fetal, childhood, and adult health. Abnormal placental function may result in a compromised pregnancy, creating pathology for the fetus and mother alike. Despite the fact that placental anatomy, function, and location has far-reaching effects for the parents and the developing offspring, ultrasound examination of the placenta is often considered secondary to the fetus by expectant parents and sonographers as well. Location, size, shape, and architecture are easily ascertained with two-dimensional techniques. Three-dimensional ultrasound and Doppler techniques have opened up the frontier of placental examination and have set the stage to make placental evaluation as fascinating as that of the fetus.

Key words: Placenta, umbilical cord, ultrasound.

Learning objectives
• To assess normal placenta by ultrasound.
• To discuss abnormal placenta and umbilical cord.
• To understand placenta in multiple gestation.

INTRODUCTION

The human placenta is a short-lived organ that is indispensable for the growth and maturation of the developing fetus. When there is normal placental function, maternal, fetal, childhood, and adult health is more common. Abnormal placental function creates pathology for the fetus and mother alike. For example, when the placental villi do not properly invade the spiral arterioles of the uterus, the placenta does not sufficiently function. Placental insufficiency, in turn, may lead to intrauterine growth restriction, oligohydramnios, maternal hypertension/pre-eclampsia, preterm delivery, or fetal death. Adult pathological conditions can be related to small placental size and suboptimal placental function. Adult onset diabetes, chronic hypertension, and obesity have also been linked to intrauterine growth restriction at birth. This is known as the Barker hypothesis. Abnormally located placentas may cause maternal morbidity and fetal compromise as a result of hemorrhage and urgent operative deliveries.

Despite the fact that placental anatomy, function, and location has far-reaching effects for the parents and the developing offspring, ultrasound examination of the placenta is not usually very interesting to most expectant parents. Examination of the placenta may be considered secondary to the fetal examination by sonographers as well. Ultrasound professionals must be cognizant of the importance of sonographic examination and documentation of the placenta. Location, size, and shape are easily ascertained with two-dimensional techniques. Three-dimensional ultrasound techniques have opened the frontier of placental examination and have set the stage to make placental evaluation as interesting as that of the fetus. The following review covers ultrasound evaluation of the normal and abnormal placenta.

ULTRASOUND OF THE NORMAL PLACENTA

Anatomy

The normal placental anatomy is comprised the umbilical cord, placental membranes and placental parenchyma. The umbilical cord has an average diameter of 0.8 to 2.0 cm and average length of 55 cm but may range from 30 to 100 cm. It is composed of one umbilical vein and two umbilical arteries surrounded by connective tissue that is gelatinous in nature called Wharton’s Jelly. The placental membranes are composed of the amnion and chorion. The amnion is first identifiable about the seventh or eighth day of embryonic development and eventually engulfs the growing embryo. As the pregnancy progresses the amnion is brought into contact with the chorion. This occurs at approximately twelve to fifteen weeks gestation. The placental
parenchyma is composed of a stromal compartment that is filled with vascular and lymphatic channels. The stroma eventually becomes slightly elevated with convex areas called lobes which are incompletely separated by grooves. The number of lobes varies from 10 to 38 and the number remains the same throughout gestation.

Size

The normally developing placenta increases in size and echogenicity as pregnancy progresses. By convention, gestational age in this chapter will be referred to as menstrual weeks and not conceptual weeks. For example, six menstrual weeks is equivalent to four conceptual weeks; since conception occurs approximately two weeks after the first day of the last menstrual period.

At approximately four menstrual weeks gestation, an intrauterine pregnancy may not be visible. Using transvaginal ultrasound, the gestational sac appears small and is fluid filled with an echogenic rim surrounding it. This represents the chorionic cavity and the implanting chorionic villi. In the early first trimester, the diameter of the gestational sac normally grows 1 mm each day. By five weeks gestation, a small mound of echogenic chorionic villi consistent with the early placenta, a yolk sac and umbilical cord may be visualized. It is at this time that the first true embryonic measurements may be taken. A normal yolk sac diameter measures between 3 and 5 mm. If the yolk sac diameter is greater than 6 mm, there is an increased risk of embryonic demise. The embryo should grow from 2 to 3 mm to 3 to 4 mm by the end of the fifth week. The embryo with cardiac activity should be visualized when the crown-rump length reaches 3 to 6 mm (approximately five to six menstrual weeks). Occasionally, cardiac activity is visualized before measurement of the crown-rump length is possible. The gestational sac diameter may increase from 16 to 23 mm by the end of the fifth gestational week. If an embryo is not visualized with transvaginal sonography by the time the gestational sac reaches 16 mm, there is a significantly increased risk of an anembryonic pregnancy.

In the first trimester, the growth of the placenta is more rapid than the fetus. After the first trimester at approximately 17 weeks, the placentatal weight increases throughout normal gestation and correlates with birth weight. As a general rule, the placental thickness in mm should approximate the gestational age in weeks plus or minus 10 mm. The extremes of placental size have been associated with abnormal pregnancy outcomes. In pregnancies at 37 weeks gestational age, the placenta should be no greater than 40 mm thick. Placental macroscopic calcifications which then increases until term. Placental calcium deposits are detected sonographically as microscopic. After 33 weeks more than half of placentas have macroscopic calcifications which then increases until term. Placental calcium deposits are detected sonographically as echogenic foci. The appearance of a grade three placenta in the late third trimester has been associated with pulmonary maturity in nondiabetic pregnancies. The amount of calcium deposition is known as the placental grade. Table 1 demonstrates the description of the placental grading system.

Grade

Ultrasound can be used to evaluate placental maturity by visualizing the changes in the intervening placental substance. Calcium deposition occurs throughout pregnancy as a normal physiologic process of placental aging. The amount of calcium deposition is known as the placental grade. Table 1 demonstrates the description of the placental grading system. In the first 2/3rd of gestation, the calcium deposition is microscopic. After 33 weeks more than half of placentas have macroscopic calcifications which then increases until term. Placental calcium deposits are detected sonographically as echogenic foci. The appearance of a grade three placenta in the late third trimester has been associated with pulmonary maturity in nondiabetic pregnancies. The clinical use of this finding is not clinically relevant with the widespread use of more accurate first and second trimesters ultrasound to date pregnancies.

There is conflicting information about the significance of grade three placentas seen prior to 34 weeks gestation. Several small studies have concluded that there is a relationship between early placental maturation and perinatal complications such as pre-eclampsia, intrauterine growth restriction and non-reassuring fetal testing. Other studies have not found such an association or disagree with the notion that advanced placental grade is associated with fetal maturation.
cigarette smoking has been associated with accelerated placental
grade.\textsuperscript{15,16} Reporting bias with regard to accurate smoking
history during pregnancy may confound placental grading
studies. There is no compelling evidence to suggest that
premature placental maturation alone should be used to guide
obstetrical decisions. The placental grade alone should not
dictate obstetrical management, instead, the entire clinical picture
should be considered.

**Shape**

The normal placental shape is a single round disk with a central
cord insertion (Fig. 1). At the end of twenty weeks, the placenta
has reached its final thickness and shape. The chorionic plate
should be the same size as the basal plate so that the fetal
membranes extend all the way to the edge. Circumferential
enlargement continues until term.

**Placental Lakes**

Placental lakes are part of the normal appearance of the placenta
in the second and third trimesters. Placental lakes may be absent,
few or numerous and seem to be more prevalent with increasing
placental thickness. Lakes are anechoic and contain maternal
blood which can be seen swirling and have low velocity venous
blood flow within them. Placental lakes have little to no clinical
significance. Some authors believe them to be precursors of
perivillous fibrin deposition or intervillous thrombosis if venous
flow decreases within the lake.\textsuperscript{17} Placental lakes are thought to
be of little clinical significance and do not seem to indicate an
increase in adverse pregnancy outcome.\textsuperscript{18}

**Subchorionic Fibrin Deposition**

Subchorionic fibrin deposition (SFD) occurs in 20% of placentas.
The SFD appear as complex cystic lesions located on the fetal
side of the placenta where fibrin deposits under the chorion
and may appear large and dramatic. With gray-scale ultrasound,
they can be mistaken for chorioangiomas. Color Doppler flow is
not seen within the mass, although low velocity swirling may
be seen with realtime ultrasound in the cystic areas.

Use of Doppler is critical to distinguish between the two.
The SFD has no clinical significance (Fig. 2).\textsuperscript{19}

**Infarcts**

Placental infarcts occur when perfusion to an area of the placenta
is reduced enough to cause necrosis of placental tissue. If
necrosis causes liquefaction or bleeding, infarcts become visible
with ultrasound. If liquefaction does not occur, infarcts may
not be seen until pathological examination. Infarcts are usually
irregularly shaped and do not contain swirling blood upon direct
visualization. Small infarcts are seen in 25% of normal
pregnancies.

If placental infarcts are large or extensive, placental function
can be compromised. Infarcts may occur as a result of maternal
vascular disease, pre-eclampsia, or poor placental implantation.\textsuperscript{20}
Perinatal morbidity is associated with infarcts of more than

\textbf{Table 1: Description of ultrasonographic placental grade}\textsuperscript{11}

<table>
<thead>
<tr>
<th>Placental grades</th>
<th>Descriptions</th>
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| Grade 0          | • No visible calcifications  
                  | • Smooth chorionic plate     |
| Grade 1          | • Scattered tiny calcifications  
                  | • Subtle indentations of chorionic plate |
| Grade 2          | • Larger basal and comma like echodensities 
                  | • Larger indentations of chorionic plate |
| Grade 3          | • Extensive basal echogenicity and circular echodensities fully outlining cotyledons 
                  | • Complete indentations of chorionic plate |

\begin{figure}[h]
\centering
\includegraphics[width=0.8\textwidth]{fig1.png}
\caption{Ultrasound of a normal placental cord insertion}
\end{figure}

\begin{figure}[h]
\centering
\includegraphics[width=0.8\textwidth]{fig2.png}
\caption{Example of subchorionic fibrin deposition. Note cystic appearance and lack of color Doppler flow}
\end{figure}
5 percent of the placental mass or greater than 3 cm in diameter. Such a placenta may be described as having a “moth eaten” appearance. Unexplained elevated maternal serum AFP has been associated with extensive placental infarction.21

ULTRASOUND OF THE ABNORMAL PLACENTA

Umbilical Cord Insertion

If the cord inserts near the edge of the placenta, this is called an eccentric insertion. A Battledore placenta refers to a cord insert on the absolute edge of the placenta, resembling a lollipop. A velamentous or membranous cord insertion refers to vessels inserting into and surrounded only by fetal membranes, with no Wharton’s jelly. A velamentous insertion may cause compromise to the integrity of the umbilical vessels because there is little support by the body of the placenta.

Umbilical Cord Vessels and Coiling

Single Umbilical Artery

The normal umbilical cord contains two arteries and one vein. The umbilical arteries branch from the fetal iliac arteries and carry less oxygenated fetal blood from the fetus to the placenta. The umbilical vein carries more oxygenated blood from the placenta to the fetus. The umbilical vein becomes the ductus venosus. After exiting the ductus venosus, blood passes into the inferior vena cava and predominately into the right atrium, foramen ovale, left atrium, left ventricle, and the fetal aorta. A smaller portion of blood from the right atrium passes into the right ventricle, pulmonary artery, ductus arteriosus, and to the fetal aorta. Single umbilical artery (SUA) occurs when one of the umbilical arteries does not form or atrophy during fetal development.

In prospective studies, SUA occurs in 1% of all deliveries and in 5% of twin deliveries. The incidence of SUA in autopsy series is twice that in prospective series. There is no evidence of a familial or genetic tendency. The incidence is dependent on race, method of cord examination, and portion of cord examined.22 The left artery is absent more commonly than the right one (70% vs 30%). The association with additional malformations appears equal for right and left in one series23 and higher if the left is absent in another series.24

The number of umbilical arteries can be ultrasonographically documented in several ways. The two arteries may normally fuse at the insertion into the placenta; therefore, the cord is best examined at the fetal insertion or in the midportion.25 The number of umbilical arteries can be determined by sagittal and transverse views of the free floating cord. In the sagittal view, two arteries must be visualized running adjacent and parallel to each other to confirm that two arteries are present. In the transverse view, two smaller arteries are visualized next to one vein. This view has the appearance of “Mickey mouse ears” (Fig. 3). Visualization of free floating cord is easier during and after the second trimester. Visualization of the number of arteries can be achieved after 11 to 12 weeks with color Doppler examination of the cord as it enters the fetus. In the transverse plane at the level of the fetal bladder, color Doppler easily maps the separation of the arteries, one on either side of the fetal bladder (Figs 4 and 5). Although high resolution ultrasound has more than at 90% sensitivity and near 100% sensitivity for detecting SUA, single artery images should be confirmed with at least to different methods of viewing and on two separate occasions to minimize the false-positive rate. The false-positive rate has been reported to be 8%.26 The presence of SUA indicates increased pregnancy surveillance and may increase parental anxiety.

The SUA has been associated with other congenital anomalies, intrauterine growth restriction, prematurity, and an
increased perinatal mortality rate when compared to infants with two umbilical arteries. In older prospective series, 20% of infants with SUA had other anomalies. A meta-analysis of 37 studies determined that there was a 66% incidence of other congenital anomalies when the diagnosis of SUA was made by examining abortuses, fetal deaths, or autopsies. More recent data support a 30 to 40% risk of another congenital anomaly, once SUA is diagnosed by ultrasound and the newborn and placenta are examined at birth.

Once SUA is identified, a thorough search for other fetal anomalies is warranted. If other anomalies are not seen, the diagnosis becomes isolated SUA. The most common associated anomalies are cardiac and renal. Although formal fetal echocardiography is recommended by many authors, a recent series suggests that it does not add more diagnostic information when the four-chamber view and outflow tracts are visualized as normal.

Table 2: Under-coiling and over-coiling of umbilical cord examined at birth and perinatal complications

<table>
<thead>
<tr>
<th>Complications</th>
<th>Odds ratio</th>
<th>95% confidence interval</th>
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<tbody>
<tr>
<td><strong>Under-coiling (UCI below 10th percentile at birth)</strong></td>
<td></td>
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<tr>
<td>Intrauterine fetal demise</td>
<td>3.35</td>
<td>1.48-7.63</td>
</tr>
<tr>
<td>Spontaneous preterm birth</td>
<td>2.16</td>
<td>1.34-3.48</td>
</tr>
<tr>
<td>Trisomy</td>
<td>5.79</td>
<td>2.07-16.24</td>
</tr>
<tr>
<td>Low 5 minute Apgar</td>
<td>3.14</td>
<td>1.47-6.7</td>
</tr>
<tr>
<td>Velamentous cord insertion</td>
<td>3.00</td>
<td>1.16-7.76</td>
</tr>
<tr>
<td>Single umbilical artery</td>
<td>3.68</td>
<td>1.26-10.79</td>
</tr>
<tr>
<td><strong>Over-coiling (UCI above 90th percentile at birth)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Asphyxia</td>
<td>4.16</td>
<td>1.30-13.36</td>
</tr>
<tr>
<td>Umbilical artery pH &lt; 7.05</td>
<td>2.91</td>
<td>1.05-8.09</td>
</tr>
<tr>
<td>Small-for-gestational age</td>
<td>2.10</td>
<td>1.01-4.36</td>
</tr>
<tr>
<td>Trisomy</td>
<td>9.26</td>
<td>2.84-30.2</td>
</tr>
<tr>
<td>Single umbilical artery</td>
<td>8.25</td>
<td>2.60-26.12</td>
</tr>
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</table>

Coiling indices have also been developed for second trimester antenatal ultrasound assessment of the umbilical cord. The antenatal umbilical coiling index (AUCI) performed in the second trimester is correlated with the postnatal umbilical coiling index (UCI). The antenatal umbilical coiling index (AUCI) is the reciprocal of the distance between two coils (AUCI = 1/ distance between two coils in centimeters). The distance between the coils is usually measured from the inner edge of an arterial or venous wall to the outer edge of the next coil along the ipsilateral of the cord. The mean AUCI in the mid trimester is 0.43. The 90th percentile is 0.602. The 10th percentile is 0.204.

Abnormal umbilical cord coiling detected by antenatal ultrasound between 18 and 23 weeks has also been associated with perinatal complications when compared to fetuses with normal coiling (small-for-gestational age 15% vs 6% and non-reassuring fetal testing in labor 25% vs 11%). There were no

Fig. 5: Color Doppler image of single umbilical artery at the level of the fetal bladder. Note the absence of the superior artery.

Umbilical Cord Coiling

Definitions of under-coiling and over-coiling of the cord have been well described for postnatal examination of the umbilical cord. The umbilical coiling index (UCI) is the number of coils divided by the total cord length. Under-coiling is defined as UCI values less than the 10th percentile. Over-coiling is defined as UCI values greater than the 90th percentile. Postnatally diagnosed coiling abnormalities (under- and over-coiling) have been associated with pregnancy complications. Fetal death, non-reassuring fetal testing, and intrauterine growth restriction occurred in 25% of fetuses with abnormal coiling. Other authors have demonstrated different types of complications associated with under- and over-coiling, respectively. Under-coiling and over-coiling of umbilical cord examined at birth and perinatal complications are shown in Table 2.
statistically significant differences with regard to 1 and 5 minutes Apgar scores, number of interventional deliveries, and meconium stained amniotic fluid in 294 patients. Documentation of umbilical cord coiling is not currently a required part of the routine ultrasound examination. After more study, it may become an important indicator of high-risk pregnancies.

**Circumvallate Placenta**

A circumvallate placenta occurs when the chorionic plate is smaller than the basal plate and the fetal membranes are folded into a circular ridge. In other words, a double layer of amnion and chorion, as well as necrotic villi and fibrin, form a white ring around the surface of the placental disk at a variable distance from the umbilical cord insertion site. Circumvallate placentas are more prone to premature separation and are associated with pregnancy complications such as placental abruption, preterm labor, and stillbirth. The accuracy of sonography for diagnosis of circumvallation appears to be limited by high false-positive and false-negative rates. A circumvallate placental edge was shown in Figure 6.

**Succenturiate Lobes**

Succenturiate lobes also known as accessory lobes are seen in almost 10% of placentas. Another synonym is a bilobed placenta and refers to a placenta with an additional lobe or lobes of placental tissue located a few centimeters away. The main lobe receives the umbilical cord insertion while placental vessels extend from and within the membrane of the main placental mass to each lobe. The location of these accessory lobes are clinically relevant as the pregnancy is at a higher risk of a vasa previa if one of the lobes is in the lower uterine segment. If connecting vessels traverse the cervical os, this is a form of vasa previa. Diagnosis and documentation is critical antenataly because if left undiagnosed rapid fetal hemorrhage and fetal death may occur at the time of ruptured membranes. These pregnancies are also at risk for retained placenta if undiagnosed antenataly. Accessory lobes usually spontaneously deliver with the main lobe due to the membranes and vessels connecting them, however, a retained placenta could occur, if accessory lobes were not diagnosed. Care should be taken not to over diagnose succenturiate lobes in placentas that are located laterally and wrap around from the anterior to posterior uterus. Succenturiate placentas are not continuous and demonstrate a significant gap in the placental body.

**Placenta Membranacea**

Placenta membranacea occurs when chorionic villi are spread all over the amniotic sac as a result of failure of regression in the first trimester. These very rare placentas are thin and may have abnormal implantation (placenta accreta) resulting in antepartum and postpartum hemorrhage. Antenatal ultrasound would show placenta covering most of the uterine cavity and reports of antenatal ultrasound diagnosis have been published. Fourteen out of twenty-six reported cases resulted in a live birth with antepartum and postpartum hemorrhage causing the highest morbidity followed by abnormal implantation.

**Molar Pregnancy**

Complete and partial molar pregnancies occur after aberrant fertilization incites abnormal proliferation of trophoblastic tissue. Normal placental villi are replaced by profuse hydropic villi. The incidence of molar pregnancy in the United States is 1/1000 pregnancies. A complete mole occurs when an empty ovum is fertilized by a single spermatozoon or two spermatozoa resulting in a 46 XX or 46 XY karyotype, in this case a fetus is not present. A partial mole is characterized by fertilization of a normal ovum by two spermatozoa resulting in a 69 XXX or 69 XXY karyotype, in this case a fetus is present. The fetus in a partial mole is almost always triploid or aneuploid and usually has multiple congenital anomalies and or growth restriction.

Ultrasound of a complete molar pregnancy demonstrates echogenic and cystic material filling the endometrium and is classically resemble a “snowstorm” appearance. High HCG levels (>100,000) and the presence of theca lutein cysts (enlarged ovaries with multiple small cysts throughout) support the diagnosis of molar pregnancy. The absence of an embryo or fetus and no amniotic fluid are also clues to the diagnosis. Sonographic features suggestive of a partial molar pregnancy include focal anechoic spaces and/or increased echogenicity of chorionic villi (Swiss cheese pattern). A fetus is present, may have cardiac activity, and is usually growth restricted. Oligohydramnios may also be present. Theca lutein cysts are absent.
Classic descriptions of the ultrasound appearance of molar pregnancies occur later in pregnancy when the patient develops bleeding or a rapidly enlarging uterus (Fig. 7). A recent study by Fowler et al found that ultrasound detected less than 50% of hydatidiform moles; detection rates were higher for complete versus partial moles and seemed to improve after 14 weeks gestation although this was not statistically significant.

Chorioangioma

Chorioangiomas are benign vascular (capillary filled) malformations that occur in 1% of placentas. Chorioangiomas range from microscopic lesion(s) that form within the placental body to large masses that protrude from the fetal side of the placenta. Masses greater than 4 to 5 cm in diameter lend an increased risk for fetal morbidity. Associated complications include output heart failure from arteriovenous shunting, platelet trapping, consumptive coagulopathy, and preterm delivery. The ultrasound appearance of a chorioangioma is round, well circumscribed, and mostly hypoechoic. There may be hyperechoic components and solid appearing areas. Generous color Doppler flow is an important distinguishing finding. Calcifications, cystic areas, and minimal color flow are more characteristic of a teratoma or subchorionic fibrin deposition both of which have little to no clinical significance. Frequent ultrasound surveillance is recommended for a chorioangiomas measuring > 5 cm to assist in detection of fetal anemia and or hydrops.

Placental Abruption

Placental abruption occurs in up to 1% of pregnancies and is defined as the premature separation of the placenta from the myometrium usually in the late second and third trimesters. Risk factors include tobacco smoking, hypertension, cocaine use, abdominal trauma, preterm premature rupture of membranes, multifetal gestation and previous abruption.

The most common location for placental separation is at the margin (edge) and may extend into a subchorionic collection. Subchorionic bleeds are more commonly described in the first and second trimesters and are usually managed expectantly. It is important to distinguish between subchorionic collections and retroplacental collections because subchorionic collections are not commonly referred to as placental abruptions and do not have the same clinical risk or connotation to obstetricians. Small subchorionic bleeds (< 15 to 60 ml) seen in the first half of pregnancy do not appear to impart adverse pregnancy outcome unlike larger bleeds. Abruptions may be partial, complete, acute or chronic and can occur in any area of the placental bed. Placental abruptions are more commonly a concern in later pregnancy when survival of the neonate becomes possible (after 24 weeks) and intervention could protect the fetus from hypoxia or death. If > 50% of the placenta appears detached, the risk of adverse pregnancy outcome is significantly increased.

The diagnosis of placental abruption is largely clinical. The utility of ultrasound in the diagnosis is poor with 50% sensitivity and thus should never be used to “rule out” a placental abruption. Clinical correlation and observation are the methods used to correctly diagnose placental abruption. However, if a retroplacental collection is noted on ultrasound, attempts should be made to quantify the amount of retroplacental hemorrhage seen. Retroplacental collections should be distinguished from the normal retroplacental complex that contains decidua, vessels, and myometrium and is usually not more than 1 to 2 cm thick in sagittal plane.

The sonographic appearance of retroplacental hemorrhage is variable. Fresh acute blood will appear hyperechoic. It becomes isoechoic in 3 to 7 days. In one to two weeks it becomes hypoechoic and after two weeks it becomes anechoic. Figures 8 and 9 are showing an example of a clinically significant retroplacental abruption.

Placenta Previa

Placenta previa generally refers to the presence of placenta lying over or near the internal cervical os. There are three types of placenta previa (complete, partial, and marginal). The low-lying placenta is not technically a previa because the placental tissue does not cover the os and is usually described as the placental edge within 2 to 3 cm of the internal os.

A complete placenta previa is diagnosed when the placenta completely covers the internal cervical os. A subgroup of complete previa includes central previas in which the cervix appears in the center of the placenta. Partial placenta previa is described when the placenta partially covers the internal os, but not completely. Marginal placenta previa is described as the placenta that only reaches the edge of the internal os.
Although transabdominal ultrasound is 95% accurate if the placenta appears well away from the lower uterine segment, there is an overall 7% false-positive rate. Transvaginal sonography is now considered the gold standard for evaluating a suspected placenta previa with an accuracy of 99%. Transvaginal sonography is performed with an empty bladder by convention which removes the confounder of the full bladder. A full bladder commonly gives the false appearance of an anterior previa during an abdominal examination. Transvaginal sonography also reduces the shadowing resulting from an engaged fetal presenting part during the third trimester and is better at evaluating the cervical involvement of a lateral or wrap around placenta. Translabial imaging is also an effective alternative. Gentle transvaginal sonography is the preferred method of detecting placenta previa. It does not appear to incite placental bleeding in the setting of placenta previa and is not comparable to a digital examination with regard to risk.

Placenta Accreta

Placenta acrreta complicates 5 to 10 percent of pregnancies with placenta previa. Placenta accreta is abnormal placentation in which the anchoring chorionic villi attach to the myometrium, rather than being contained by decidua. The normal decidua basalis and fibrinoid layer (Nitabuch's layer) are defective and as a result the placental villi adhere to the myometrial layer and may penetrate through it to reach other structures. There are three levels of invasive placentas. The first is accreta where the placenta adheres to the fetal surface of the myometrium. The second is increta where the placenta invades into the myometrium. The third is placenta percreta where the placenta invades all the way through the myometrium and often into the bladder wall and or into the bladder cavity itself if the placenta is anterior. Abnormal placentation may also occur posteriorly which is not as common and more difficult to diagnose by ultrasound. All forms of placenta acrreta are associated with significant antenatal and/or intrapartum hemorrhage at the time of placental separation/removal.

Ultrasound is 85% sensitive for the detection of placenta accreta in high-risk women. The normal hypoechoic placental-myometrial interface is obscured and the placenta appears continuous with the myometrium. Figure 10 shows a normal placental-myometrial interface. Color Doppler is also sensitive (82%) and specific (97%) for the diagnosis of placenta previa/accreta when four criteria are used. The criteria are diffuse and focal intraparenchymal placental lacunar flow, hypervascularity of the bladder and uterine serosa, prominent subplacental venous complex and loss of subplacental Doppler vascular signals. Hematuria may also support the diagnosis of placenta percreta. Figure 11 for gray-scale image of placenta percreta as well as example of a previa. Figure 12 shows color Doppler of placenta percreta.

Although transabdominal ultrasound is 95% accurate if the placenta appears well away from the lower uterine segment, there is an overall 7% false-positive rate. Transvaginal sonography is now considered the gold standard for evaluating a suspected placenta previa with an accuracy of 99%. Transvaginal sonography is performed with an empty bladder by convention which removes the confounder of the full bladder. A full bladder commonly gives the false appearance of an anterior previa during an abdominal examination. Transvaginal sonography also reduces the shadowing resulting from an engaged fetal presenting part during the third trimester and is better at evaluating the cervical involvement of a lateral or wrap around placenta. Translabial imaging is also an effective alternative. Gentle transvaginal sonography is the preferred method of detecting placenta previa. It does not appear to incite placental bleeding in the setting of placenta previa and is not comparable to a digital examination with regard to risk.

![Fig. 8: Ultrasound of a patient with a clinical abruption with accompanying preterm labor. Note the significant funneling of the cervix and fetal caput](image)

![Fig. 9: The same patient with a placental abruption. Note the echolucent area posterior to the fundal placenta](image)

![Fig. 10: Ultrasound of a normal placental-myometrial interface. Note the thin homogeneous hypoechoic line indicating a normal Nitabuch’s layer](image)
A high index of suspicion by the sonographer could be life-saving for women with risk factors. Risk factors for placenta accreta include prior uterine surgery, placenta previa, maternal smoking, and advanced maternal age. The incidence of placenta accreta among women with one, two and four or more cesarean sections and a concomitant placenta previa is 25, 47, and 67%, respectively.

Multiple Pregnancy Placentaion

One of the most important reasons for performing ultrasound for multiple pregnancies is to determine the number of amnions and the number of chorions (placentas). Amnioncicy and chorionicity are related to pregnancy outcome and will determine obstetrical management. Diamniotic dichorionic twin pregnancies have the lowest mortality rate among twins (9%). They are followed by monochorionic diamniotic (26%) and monoamniotic (up to 50%). Conjoined twins have the highest mortality rate (75%). Inpatient admission, antenatal steroid administration, and early delivery at 32 to 34 weeks significantly reduce perinatal mortality in monoamniotic twins. In one series, monoamniotic twins managed with outpatient fetal monitoring and early delivery had a perinatal mortality rate of 14.8%. Patients managed as inpatients with daily fetal monitoring and early delivery had a perinatal mortality rate of zero. Twin-twin transfusion syndrome and unequal placental sharing are thought to contribute most to the increased mortality for monochorionic twins. Cord entanglement and compression is the major risk for monoamniotic twin pregnancies (Figs 13 and 14).

Fig. 11: Ultrasound of placenta percreta. Note cystic lacunae and absence of echogenic layer between placenta and myometrium. Placenta previa is also seen. This patient had a history of 4 prior cesarean deliveries.

Fig. 12: Color Doppler image of a placenta accret/percreta. Note color flow mapping of placental vessels through the myometrium and inside the maternal bladder.

Fig. 13: Ultrasound of cord entanglement in monoamniotic twins at 29 weeks gestation. Note the two loops of one cord wrapped around the other cord. See the next image for the photograph of the actual cord at delivery.

Fig. 14: The same patient (Fig. 13). Significant cord entanglement at delivery in monoamniotic twins. This patient underwent intensive inpatient fetal monitoring beginning at 25 weeks. Two vigorous infants were electively delivered at 32 weeks.
Two-thirds of twin pregnancies in the United States are dizygotic or fraternal (two ova fertilized by two sperms). Dizygotic pregnancies are always diamniotic dichorionic. One-third of twin pregnancies in the United States are monozygotic or identical. On very rare occasions, post-zygotic mutations can cause genetic discordance among monozygotic pairs. All monochorionic twin pregnancies are monozygotic, but not all monozygotic pregnancies are monochorionic. For monozygotic pregnancies (one ovum fertilized by one sperm), the timing of embryonic splitting determines the number of amnions and chorions. Once the chorion, amnion, or embryo differentiates, it cannot split. The chorion differentiates first (day 4 to 8) followed by the amnion (day 8 to 13). Table 3 presents timing of splitting and its relationship to number of amnions and chorions.

Patients with twins during ultrasound often ask, “are my babies identical?” The answer is based on one of three scenarios. If monochorionicity is documented then the answer is “yes with very rare exception”. If dichorionicity is present and the genders are different then the answer is “no”. If dichorionicity is present and the genders are the same the answer is “possibly”. There is an 8% chance that same gender dichorionic gestations will be identical. In the third scenario, newborn zygosity testing (HLA matching or DNA polymorphism analysis) is the only way to prove identical twins. Zygosity testing is rarely clinically indicated. Ultrasound examination is most helpful for determining chorionicity, not zygosity.

Table 3: Chorionicity and amnionicity are determined by timing of embryo splitting

<table>
<thead>
<tr>
<th>Types of twinning</th>
<th>Timing of embryo splitting</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dichorionic diamniotic</td>
<td>Less than 4 days (2-8 cell stage)</td>
</tr>
<tr>
<td>Monochorionic diamniotic</td>
<td>4-8 days (blastocyst stage)</td>
</tr>
<tr>
<td>Monochorionic monoamniotic</td>
<td>8-13 days (early embryonic disk)</td>
</tr>
<tr>
<td>Conjoined</td>
<td>Greater than 13 days (later embryonic disk)</td>
</tr>
</tbody>
</table>

The presence or absence of the dividing membrane determines amnionicity. Chorionicity can be ultrasonographically distinguished in several ways. Simply documenting the presence or absence of a dividing membrane is insufficient information. Sonographers should make an effort to determine number of chorions as well as the number of amnions. Discordant gender (male and female) is diagnostic for dichorionicity. A thick dividing membrane, triangular membranes at the placental insertion (lambda sign or twin peak sign), visualization of four membrane layers, and separate placentas also indicate dichorionicity.

A monochorionic membrane appears subjectively like a strand of hair (Fig. 15). An absent membrane indicates monoamnioncity (Fig. 16). A dichorionic membrane is easily visible (Fig. 17). A membrane thickness of 2 mm or greater correctly identifies dichorionic pregnancies in 80 to 90% of...
cases. Accuracy can be improved to 100% by visualizing 4 layers of membrane. Free floating membranes may give a false appearance of multiple layers; therefore, membrane layers should be examined near the insertion site only. The twin peak or lambda sign occurs when chorionic villi fill the triangular interchorionic space at the placental insertion site (Fig. 17). Monochorionic gestations have only two membranes layers. They cannot create the interchorionic space; therefore, villi cannot fill a nonexistent space. The insertion of a monochorionic membrane resembles a “T” instead of a “λ” and is known as the T sign (see Fig. 15).

The first trimester is the optimal time to document chorionicity with ultrasound. The presence of a single yolk sac and two embryos is also diagnostic for monochorionicity. The first trimester dichorionic membrane appears at it is thickest and is clearly visualized. The placental membrane insertion (lambda sign) is more prominent in the first trimester and may become less prominent as pregnancy progresses. Visualization of the lambda sign is nearly 100% accurate for prediction of chorionicity. At 10 to 14 weeks, twin pregnancies with the lambda sign can be classified as dichorionic. Pregnancies with absent lambda sign can be classified as monochorionic. At 10 to 14 weeks the lambda sign is seen in 100% of dichorionic gestations with fused placentas and 90% of dichorionic gestations with separate placentas. At 16 to 20 weeks, the lambda sign is seen less often in dichorionic pregnancies (75% with separate placentas and 93% with fused placentas). The absence of the lambda sign does not exclude chorionicity during the second trimester. The presence of separate placentas alone is not diagnostic of chorionicity. Separate placental masses may occur in 3% of monochorionic gestations. To reduce diagnostic error, documentation of multiple markers of chorionicity is recommended. In prospective studies using first and second trimester ultrasounds, multiple markers of chorionicity have an overall accuracy of 95 to 99%.

First trimester ultrasounds that incorporate multiple markers have accuracy closer to 99%. Multiple ultrasound markers of chorionicity should also be applied to higher order multiples. Membranes, membrane insertions, and placentas should be documented for all of the sacs. High order multiples can have any combination of chorionicity particularly if conceived with assisted reproductive techniques. Any combination is possible with early fetal demise and embryo splitting. Documentation of chorionicity is extremely important before considering multifetal reduction. Multifetal reduction is contraindicated in multiple pairs that appear monochorionic, unless the desired effect is the reduction of both fetuses in that pair.

**PLACENTAL 3D DOPPLER ULTRASOUND**

Uteroplacental development is dependent on invasion of the spiral arteries by trophoblast to create a low resistance system.
appear to increase slowly and steadily as pregnancy advances. Three-dimensional color Doppler of placental vasculature is in its infancy. It is the hope that future investigation will prove this technique clinically useful.

CONCLUSIONS

Ultrasound examination of the placenta is just as important as the examination of the fetus. When examining the placenta, sonographers and sonologists have a unique opportunity to detect problems that will significantly affect perinatal outcome. Two-dimensional imaging provides excellent information about the location and architecture of the placenta. Three-dimensional Doppler imaging of the placenta is emerging as a more detailed method of examining uteroplacental structure and function.

REFERENCES


