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Ultrasound abnormalities of the amniotic fluid, membranes, umbilical cord, and placenta

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The advent of prenatal ultrasound has not only allowed clinicians to obtain more information about fetal anatomy, but also about the intrauterine environment through the evaluation of the amniotic fluid volume, fetal membranes, the umbilical cord, and the placenta. Evaluation of these entities is an integral part of every sonographic evaluation. This article reviews various conditions that can be detected by prenatal ultrasound evaluation.

Amniotic fluid

Sonographic evaluation of the amniotic fluid volume

The amniotic fluid volume is the sum of the inflows and outflows of the amniotic sac and is a reflection of the intrauterine environment. In early gestation, before the development of fetal urination and swallowing, the amniotic fluid is likely formed by active transport by the amnion into the amniotic space and water is allowed to flow passively [1]. In later gestation, when the fetal skin is keratinized, the major pathways include fetal urination, fetal swallowing, fetal lung fluid secretion, and intramembranously [1].

Ultrasound visualization of the amniotic fluid permits both subjective and objective estimates of the amniotic fluid volume. Examination of the amniotic fluid volume has become an integral part of both routine and targeted ultrasound. Subjective evaluation of the amniotic fluid volume is usually performed in pregnancies less than 20 weeks gestation; however, the use of a numerical estimate provides a more accurate assessment of fluid volume over time, allowing comparisons on follow-up. Normal amniotic fluid volumes have been defined across gestational age with a progressive increase from 8 weeks gestation to a peak of

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800 mL at 32 weeks gestation, followed by a slow decline to term and beyond [2]. Using dye dilutional studies, Didly et al [3] showed a correlation between previously reported amniotic fluid indices and term pregnancy volumes [2].

Abnormalities of the amniotic fluid volume have been associated with adverse perinatal outcome and may be a marker for other fetal abnormalities, such as congenital malformations, aneuploidy, and growth restriction. For example, oligohydramnios in the absence of premature rupture of membranes can be associated with urinary tract abnormalities, such as renal agenesis.

Several ultrasound techniques have been described to estimate the amniotic fluid volume. In 1984, Chamberlain et al [4] introduced the concept of using the depth of the maximum vertical pocket. This semiquantitative estimate measured the deepest pocket of amniotic fluid free of umbilical cord or fetal parts in the anteroposterior plane of the uterus. The amniotic fluid volume was considered normal if the maximum vertical pocket was greater than 2 cm and less than 8 cm. Oligohydramnios was defined as a pocket less than 1 cm in depth and polyhydramnios was defined as a pocket over 8 cm. In subsequent studies, the single deepest pocket technique was shown to have several shortcomings. The amniotic fluid index (AFI) was introduced as a more reliable estimate of the amniotic fluid volume [5]. This technique involves dividing the uterus into four quadrants summing the deepest vertical pockets free of umbilical cord or fetal parts. The normal range of AFI in a population of patients at increased risk for poor perinatal outcome and already undergoing antenatal testing was defined as greater than 8 and less than 18 cm [5].

Moore and Cayle [6] established normal limits of AFI per week of gestation in normal pregnancy. Oligohydramnios (5th percentile) was defined as less than 7 cm and polyhydramnios (95th percentile) greater than 21 cm. An AFI of less than or equal to 5 cm was seen in less than 1% of normal term patients and an AFI of greater than 18 cm was seen in 15% of the normal population. Magann et al [7] introduced the two-diameter semiquantitative measurement of the amniotic fluid volume where the vertical depth of the maximum vertical pocket is multiplied by the largest horizontal diameter again free of umbilical cord or fetal parts. Recently, this group performed a large-scale study comparing the AFI, single deepest pocket, and two-diameter pocket in normal pregnancies. They concluded that the AFI was the most acceptable method for assessing fluid status in a singleton gestation [8].

Disorders of the amniotic fluid volume

Oligohydramnios complicates 0.5% to 8% of pregnancies and the prognosis for pregnancies complicated by oligohydramnios is gestational age–dependent. Fetal urination is a major source of amniotic fluid in the second half of pregnancy and any condition preventing formation of urine or entry into the amniotic sac results in oligohydramnios. In a series of 128 fetuses with severe oligohydramnios in the mid-trimester (13 to 24 weeks gestation), fetal abnormalities were detected in 51%, premature rupture of membranes in 34%, abruption in 7%, and

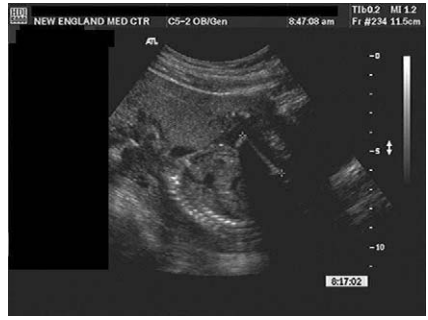

Fig. 1. Anhydramnios at 19 weeks gestation. There is no measurable pocket of fluid. The fetus is in close approximation to the placenta.

growth restriction in 5%. Aneuploidy was found in almost 1% of anomalous fetuses and in only 4% of cases no cause was detected [9].

The diagnosis of oligohydramnios is obtained by ultrasound evaluation of an AFI less than 5 cm at term (greater than 2 standard deviations below the mean); less than 8 cm before term; or a fluid pocket less than 2 cm (Figs. 1 and 2) [7]. Oligohydramnios with intact membranes warrants a comprehensive evaluation to detect possible fetal and placental abnormalities, growth restriction, or aneuploidy. Indigo carmine dye injected into the amniotic fluid cavity may facilitate the diagnosis of rupture of membranes if dye is seen on a tampon inserted into the vagina. Elevated maternal serum alpha-fetoprotein levels have also been linked to oligohydramnios, intrauterine growth restriction, preterm delivery, and fetal demise [10].

Several studies have correlated the AFI with perinatal outcome [4,11–13]. Chamberlain et al [4] reported a 13-fold increase in perinatal mortality if the amniotic fluid volume was marginally decreased and a 47-fold increase when severe oligohydramnios was present. The most common high-risk factors associated with oligohydramnios are intrauterine growth restriction and postterm pregnancy [4,12]. In a meta-analysis in 1999, an antepartum and intrapartum AFI of less than 5 cm was associated with an increased risk of cesarean section delivery for

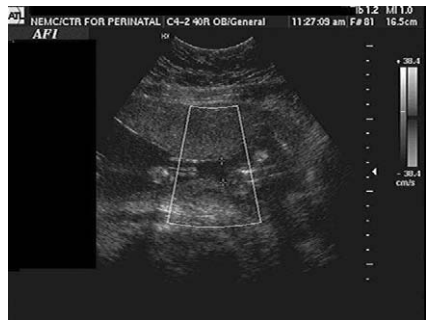

Fig. 2. Oligohydramnios. One pocket of fluid measuring less than 2 cm.

nonreassuring fetal heart rate tracings and Apgar scores of less than 7 at 5 minutes [11].

The addition of color flow Doppler has been reported to decrease significantly the measured AFI and increase the diagnosis of oligohydramnios [14,15]. The question of whether color Doppler should be used routinely remains controversial because the current normograms were obtained without the use of color Doppler.

Polyhydramnios

Polyhydramnios is defined as an AFI greater than the 95th percentile for gestational age or a maximum vertical pocket greater than 8 cm (Fig. 3) [5,6,8]. Polyhydramnios complicates approximately 1% of all pregnancies. Ultrasound evaluation of the amniotic fluid allows polyhydramnios to be classified as mild if the maximum pocket is between 8 and 11 cm, moderate if the maximum pocket is 12 to 15 cm, and severe if the maximum pocket is over 16 cm [16]. The latter occurs in less than 5% of all cases of polyhydramnios. The degree and prognosis of polyhydramnios is related to the underlying etiology. When a diagnosis of polyhydramnios is made, careful evaluation of the fetal anatomy is warranted. Fetal abnormalities of the central nervous system, gastrointestinal tract, and musculoskeletal system have been reported [16,17]. Because fetal swallowing is an important mechanism in controlling the AFI, such abnormalities as duodenal or esophageal atresia are often associated with increased fluid volume (Fig. 4). Polyhydramnios is seen in 35% of cases of anencephaly [18]. The possible pathogenesis includes transudation of the exposed meninges and lack of antidiuretic effect because of impaired arginine vasopressin secretion.

Although many cases of polyhydramnios are idiopathic, when a cause is found almost 80% have moderate or severe polyhydramnios [19]. Idiopathic polyhydramnios, usually in the mild range, although associated with macrosomia and cesarean delivery, has not been associated with adverse perinatal outcome [19]. Maternal diabetes, fetal infection, aneuploidy, and multiple gestations have also been associated with polyhydramnios [17–19]. Polyhydramnios that develops secondary to maternal diabetes is less well understood. Maternal hyperglycemia

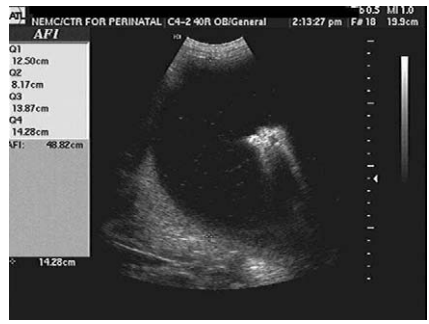

Fig. 3. Amniotic fluid pocket measuring 14.8 cm consistent with polyhydramnios.

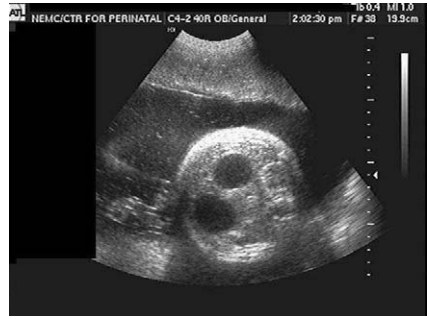

Fig. 4. Double bubble sign associated with duodenal atresia in a fetus with polyhydramnios.

causes fetal hyperglycemia, which may lead to osmotic diuresis, increased glomerular filtration rate, and urinary output [1].

Amniotic fluid volume in multiple gestations

Estimating the amniotic fluid volume in multiple gestations can be challenging because of the irregularity of the cavities occupied by each fetus and the ability to locate the separating membrane. Magann et al [20] compared the AFI, maximum vertical pocket, and two-diameter pockets in 45 dichorionic-diamniotic twin gestations where dye had been injected into each sac. When the AFI was normal, all three techniques were equivalent.

Membranes

Amnion rupture sequence or amniotic band syndrome

The amnion rupture sequence, commonly known as ''amniotic band syndrome,'' is a cause of fetal deformations involving the limbs, trunk, and craniofacial region. Most cases are sporadic with reported incidence ranges from 1 in 1200 to 15,000 live births [21]. This discrepancy in incidence rates is likely caused by misdiagnoses. The clinical manifestations of amniotic band syndrome vary from minor deformities, such as syndactyly, to severe and even lethal anomalies [22].

Several theories have been proposed to explain the occurrence of these anomalies [23–25]. The amnion and chorion normally fuse by 14 weeks; however, separation may persist into the second trimester and may be a normal finding. Persistence after 16 weeks may be associated with amnion rupture, subchorionic bleed, and early amnion rupture sequence. Rupture of the amnion without rupture of the chorion leading to transient oligohydramnios and passage of the fetus from the amniotic to the chorionic cavity is one of the most widely accepted theories [23]. The variable phenotype seen with amniotic band syndrome has been attributable to the timing of the rupture. Early rupture, within

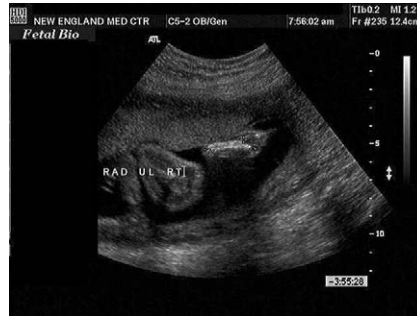

Fig. 5. Amputation of the fetal hand from suspected amniotic band syndrome.

45 days of gestation, leads to the most severe malformations, particularly of the central nervous system, face, and viscera. Amniotic bands may tear or disrupt previously normally developed structures leading to amputations and nonanatomic facial clefts (Fig. 5) [25].

Amniotic band syndrome may be detected sonographically by demonstrating fetal deformities in a nonembryologic distribution or by the visualization of bands; the latter may be extremely difficult [22,25]. The appearance of sheets or bands of amnion attached to the fetus with resultant deformity or restriction of motion allows an accurate diagnosis to be made (Fig. 6). Cranial involvement may be detected as anencephaly and facial clefts; visceral involvement may result in omphalocele or bladder exstrophy; and various limb deformities, such as constriction rings, lymphedema, amputations, and clubfoot, may occur [25]. The most common defect is constriction bands of the extremities [25]. Constriction of the umbilical cord and subsequent fetal demise has also been reported [22].

The antenatal course is dependent on the nature of the lesions and extent of the malformations [24,25]. Management may depend on the severity of the sonographic findings, and includes expectant management or termination of pregnancy. Amniocentesis should be offered if the diagnosis remains unclear. Crombleholme et al [21] reported on fetal intrauterine intervention in the lamb model with release of constrictive lesions.

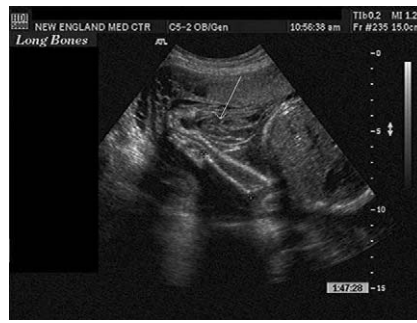

Fig. 6. Note the presence of amniotic sheets in the upper right corner (*arrow*).

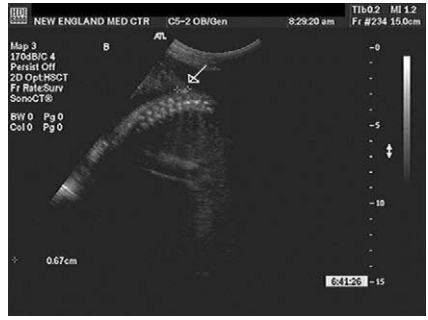

Fig. 7. Amnion nodosum. Small echogenic nodule along the membranes of a donor twin in a case of twin-to-twin transfusion syndrome (*arrow*).

Amnion nodosum

Amnion nodosum are nodules seen in the amnion that on pathologic review are often called squamous amnionic metaplasia. These nodules can vary in size from 1 to 5 mm in diameter and are composed of ectodermal debris, including vernix, hair, squames, and sebum. They are associated with oligohydramnios and are most commonly found in fetuses with renal agenesis, prolonged premature rupture of membranes, or the placenta of a donor twin in twin-to-twin transfusion syndrome (Fig. 7). The clinical significance of amnion nodosum is unknown.

Umbilical cord

The sheathing of the body stalk and omphaloenteric duct forms the umbilical cord during the embryonic period by the amniotic somatopleura. Sonographically, the umbilical cord can be seen as early at 42 days gestation and is well established by 8 to 9 weeks (Fig. 8). Transvaginal evaluation has enabled visualization of the physiologic herniation of the midgut, which occurs between

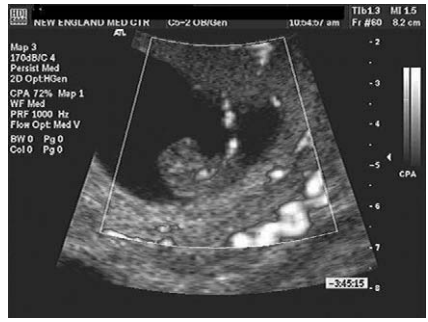

Fig. 8. Umbilical cord in the first trimester.

Table 1
Abnormalities of the umbilical cord

Abnormal length
Absence of the umbilical cord
Abnormal cord insertion
Marginal insertion
Velamentous insertion
Vasa previa
Distortional abnormalities
Loops
Knots
Coiling
Entanglement
Abnormalities of vessel number
Single umbilical artery
Persistent right umbilical vein
Umbilical cord masses and vascular malformations
Cysts
Umbilical cord hematoma
Umbilical vein or artery thrombosis
Umbilical vein varix
Umbilical artery aneurysm

Modified from Abnormalities of the umbilical cord. In: Bianchi DW, Crombleholme TM, D'Alton ME, editors. Fetology. New York: McGraw Hill; 2000; with permission.

6 and 10 to 12 weeks gestation. The umbilical cord is composed of three vessels: two arteries and one vein arranged in a spiral or helical fashion within the cord. It also contains specialized mucopolysaccharide-rich mesenchyme known as "Wharton's jelly" that protects the cord from compression. Abnormalities in the number of vessels including a two-vessel cord and persistent right umbilical vein have been reported (Table 1) [26–28].

The sonographic evaluation of the umbilical cord includes the number of vessels, the observation of coiling and looping of the cord, and Doppler velocimetry studies. The cord is evaluated at the fetal insertion site along the fetal abdominal wall, at the placental insertion site, and at a segment floating in the amniotic fluid.

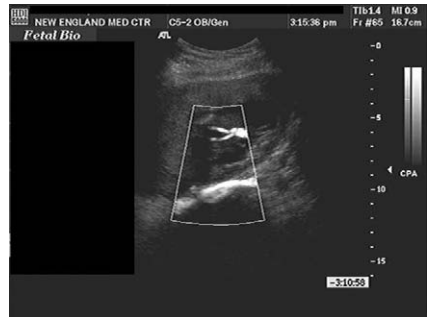

Fig. 9. Normal umbilical cord with Doppler color showing two umbilical arteries around the fetal bladder.

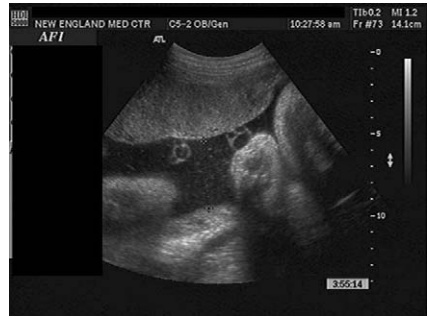

Fig. 10. Normal umbilical cord in cross-section demonstrating three vessels. The umbilical vein has the larger diameter.

A normal cord with two umbilical arteries can be confirmed on transverse section by visualizing two vessels lateral to the fetal bladder (Figs. 9 and 10).

The umbilical cord grows by tension as a result of fetal movement. The mean length of a term umbilical cord is 60 cm. A short cord is defined as less than 35 cm at term [28]. Measurement of the umbilical cord by sonographic evaluation, however, is not routinely performed.

Coiling of the umbilical cord is believed to provide protection against forces, such as tension, compression, and entanglement. Although up to 30% of umbilical cords are uncoiled at 20 weeks gestation, less than 5% lack vascular coiling at term [29]. Uncoiled umbilical cords have been associated with increased perinatal morbidity and mortality including intrauterine growth restriction, oligohydramnios, fetal anomalies, preterm delivery, and fetal demise [30].

Knots of the umbilical cord are classified as true or false knots. Prenatal diagnosis of true and false knots is extremely challenging, because there are no typical prenatal sonographic characteristics. Rarely, a vascular protuberance along the cord can be seen with false knots [31].

The umbilical cord frequently becomes coiled around fetal parts, particularly the neck, termed a ‘‘nuchal cord.’’ Sonographic detection was first reported by Jouppill and Kirkinen [32]. Nuchal cords may be present in 25% of pregnancies; however, a single nuchal loop is most likely an incidental finding not associated with fetal morbidity and mortality. The incidence of perinatal death secondary to a nuchal cord is very low. The presence of multiple nuchal cords has been associated with moderate to severe variable deceleration while monitored in labor, meconium-stained amniotic fluid, need for resuscitation, and lower umbilical artery pH [33]. Sensitivity with color Doppler to detect a nuchal cord is over 80%, which is higher than conventional gray-scale ultrasound [34].

Nyberg et al [35] reported the first case of ultrasonographic evidence of cord entanglement in a monochorionic-monoamniotic twin gestation. Ultrasound evaluation and color Doppler notes a mass-like structure between the two fetuses. Each umbilical cord should be traced to each of the twins (Fig. 11).

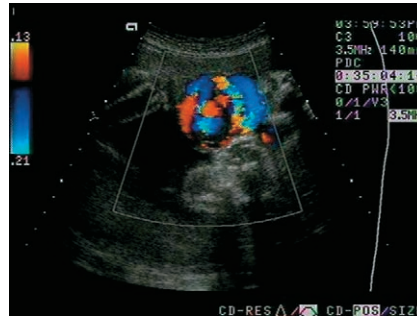

Fig. 11. Mass of entangled umbilical cords in a monochorionic-monoamniotic twin gestation.

Single umbilical artery

Single umbilical artery (SUA) is one of the most common congenital abnormalities with an incidence of about 1% of all pregnancies. Possible mechanisms giving rise to a SUA include primary agenesis of one artery, atrophy or atresia of a previously present artery, and persistence of single alloantoic artery. The left umbilical artery is more commonly absent [26]. SUA is a developmental abnormality with no known recurrence risk.

Ultrasonographic imaging has permitted the prenatal diagnosis of a SUA and a cross-section of the umbilical cord has become an integral part of every prenatal sonogram. The infrarenal portion of the umbilical arteries can be seen on transverse section lateral to the fetal bladder. Absence of one of the arteries confirms a SUA (Figs. 12 and 13). Diagnosis of a SUA has been associated with increased perinatal morbidity and mortality mostly because of an association with congenital malformations, the incidence of which may be 30% to 60% [26–28]. A SUA can be associated with malformations of almost any major organ system. The association of a single umbilical artery and other congenital abnormalities warrants a targeted sonogram and possibly a fetal echocardiogram. As an isolated finding, SUA has not been an association with aneuploidy; however, in the

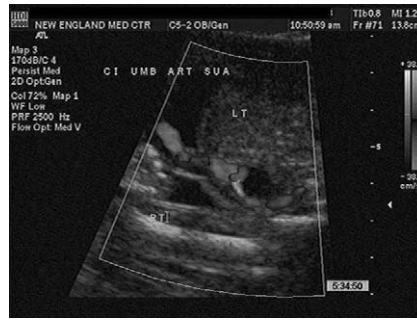

Fig. 12. Color Doppler demonstrating absence of one umbilical artery.

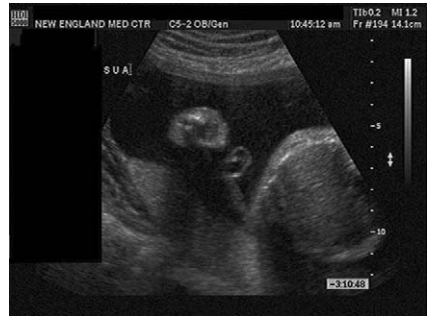

Fig. 13. Cross-section of the cord showing two vessels.

presence of other abnormalities the reported incidence of aneuploidy has been as high as 8% [26,27]. Fetal growth should be assessed in the third trimester in cases of isolated SUA because growth restriction has been reported [28].

Abnormalities of cord insertion

The umbilical cord inserts at or near the center of the placenta in over 90% of cases. Abnormalities of cord insertion can be detected sonographically and may be clinically important. Marginal insertion, also referred to as the "battledore placenta," occurs when the cord inserts at the placental margin. Marginal insertion can be seen in 5% to 7% of term pregnancies.

Velamentous insertion occurs in 1% to 2% of term singleton pregnancies and more frequently in multiple gestations. The umbilical vessels separate into the membranes at a distance from the placental margin surrounded only by a fold of amnion devoid of Wharton's jelly. Clinically, velamentous cord insertion has been associated with cord compression, poor fetal growth, thrombosis, placenta previa, and vas previa (Fig. 14).

In vasa previa, some of the fetal vessels are seen in the membranes crossing the region of the internal os ahead of the presenting part. Risk factors for vasa previa include velamentous insertion, succenturiate lobe, and low-lying placenta

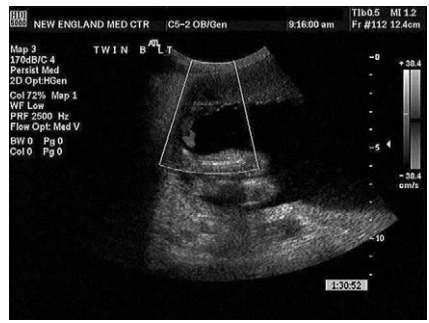

Fig. 14. Velamentous insertion of the umbilical cord.

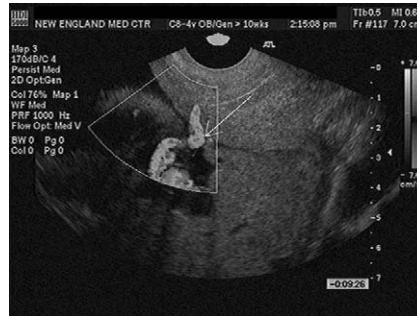

Fig. 15. Color Doppler demonstrating the presence of fetal vessels above the internal os (*arrow*).

[36]. Vasa previa can be detected by ultrasound evaluation and detection can significantly decrease fetal mortality [36]. Potential danger of exsanguination exists if rupture of membranes causes a nick in the fetal vessels. Transvaginal color Doppler imagining has increased the ability to diagnose cases of vasa previa in the mid-trimester (Fig. 15) [37].

Abdominal wall defects and the umbilical cord

Gastroschisis, omphalocele, and body stalk anomaly are abdominal wall defects that are related to the development of the umbilical cord. Gastroschisis is an abdominal wall defect likely secondary to a vascular abnormality resulting in a right paraumbilical defect. The umbilical cord is normally inserted with herniation of the gut to the right without a membranous coating. Omphalocele is distinguished from gastroschisis because the cord insertion is seen at the apex of the membrane that covers the abdominal wall defect. Body stalk anomaly is the most severe abdominal wall defect that results in the absence or shortening of the umbilical cord. The abdominal organs lie outside the abdominal cavity and appear attached to the placenta. The proposed causes of this complex abnormality include resemblance to the amnion rupture sequence or a vascular disruption and nonclosure of the abdominal wall [25,38]. The pattern of anomalies depends on the degree of abnormal development of the four embryonic folds. Sonographic evidence of body-stalk anomaly is suspected in the presence of large thoracic or abdominal wall defect; skeletal abnormalities, such as kyphosis or scoliosis; and absent or very short umbilical cord [25,38]. When the diagnosis is made, this anomaly is uniformly fatal.

Umbilical cord masses

Umbilical cord masses include cysts, tumors, aneurysms, and varices. The presence of these masses warrants careful evaluation of the umbilical vessels to evaluate complications, such as cord compression or thrombosis, and a detailed fetal survey for possible associated fetal malformations or compromise.

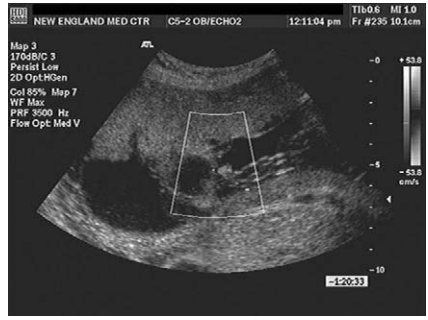

Fig. 16. Umbilical cord cyst.

Umbilical cord cysts may be true cysts or pseudocysts and this differentiation can only be established by pathologic evaluation. True cysts are derived from the embryonic remnants of either the allantoic or omphalomesenteric duct and are more common toward the fetal end of the cord (Fig. 16). A prospective screening study of 859 women noted an incidence of 3.4% in the first trimester [39]. Although many of these resolved, over 20% of these umbilical cord cysts persisted into the second and third trimester and were associated with fetal aneuploidy or fetal structural defects [39]. The detection of umbilical cord cysts in the second trimester warrants the offering of fetal karyotyping.

Prenatal sonographic diagnosis of an umbilical cord varix has been reported. Although rare, varicosity of the umbilical vein may occur in the intra-amniotic portion of the umbilical vein and the fetal intra-abdominal portion. The intra-amniotic portion is seen as an abnormal dilation of the vein at the abdominal insertion site that can lead to venous compression. The extrahepatic portion of the fetal intra-abdominal umbilical vein has been measured in normal fetuses and found to increase throughout gestation from 3 mm at 15 weeks gestation to 8 mm at term [40]. The varix of the fetal intra-abdominal umbilical vein results in an oval cystic mass between the abdominal wall and the inferior edge of the fetal liver. The detection of venous flow with color Doppler distinguishes a varix from other types of masses (Figs. 17 and 18) [40].

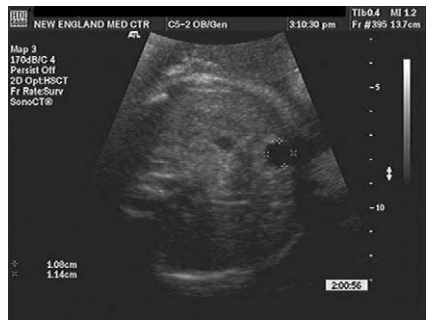

Fig. 17. Umbilical varix of the fetal intra-abdominal portion.

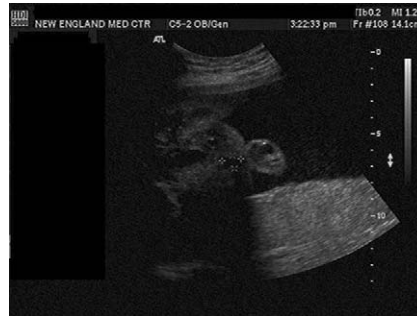

Fig. 18. Varix of the intra-amniotic portion of the umbilical cord.

The cause of the fetal intra-abdominal umbilical vein remains unknown. Possible etiology includes dilation caused by an intrinsic weakness in the wall of the extrahepatic portion of the umbilical vein, possibly of the portion where the right umbilical vein becomes obliterated during embryogenesis [28,40]. Mahoney et al [40] suggested that the detection of an umbilical cord varix is associated with an increased risk of adverse fetal outcome including fetal demise necessitating antenatal monitoring.

Spontaneous umbilical cord hematoma is a very rare condition and most cases are iatrogenic following cordocentesis or more rarely amniocentesis [31]. These usually appear as focal masses and can be associated with a 50% risk of fetal loss. Hemangiomas of the umbilical cord are the most common tumors of the umbilical cord albeit rare entities. These are most commonly seen at the placental insertion of the umbilical cord and sonographic appearance may be that of an echogenic or multicystic mass with color Doppler [31].

Placenta

The placenta can be identified as early as 6 weeks gestation by transvaginal evaluation and by 10 weeks gestation by transabdominal evaluation as an echogenic and thickened rim around the gestational sac [41]. It is distinct from the hyperechoic myometrium. The placenta increases in size throughout gestation and typically has a discoid shape. Hypoechoic areas known as ‘‘venous lakes’’ may be present in the placental parenchyma and on color Doppler blood flow are usually absent.

Although placental volume in the second trimester may be a predictor of fetal outcome, there is no accurate or acceptable method of measurement [42]. The thickness of the placenta can be assessed on sonogram and rarely exceeds 4 cm. Hyperplacentosis or placentomegaly has been associated with several entities including diabetes mellitus; immune and nonimmune hydrops; fetal infections, such as parvovirus and syphilis; molar pregnancy; and aneuploidy [41].

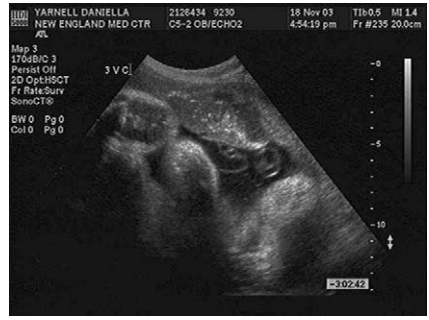

Fig. 19. Calcifications in the placenta.

Calcium deposition also occurs normally in the placenta and appears sonographically as bright intraplacental echoes. Numerical grade has been assigned from grade 0 to grade III, where grade 0 is no calcifications and grade III has extensive echogenicity. Placental grading was believed to correlate with fetal lung maturity; however, larger studies have shown this was not reliable and amniocentesis for fetal lung maturity studies remains the gold standard [43]. Placental grading has little clinical significance, although cigarette smoking has been associated with increased calcifications (Fig. 19) [44].

Placental shape abnormalities

Abnormalities of placental shape are most often secondary to disappearance of villi. The placenta normally develops where the chorionic villi interfacing the decidua basalis grow and the remaining villi undergo atrophy. Placental abnormalities can be detected by sonogram and may affect clinical management and obstetric outcome.

Placenta membranacea is an uncommon condition with an incidence of 1:3000 live births. With placenta membranacea, all the fetal membranes are covered by functioning villi. The placenta develops as an abnormally thin membranous structure. On ultrasound evaluation, the placenta is seen over the entire uterine surface. Clinically, placenta membranacea can be associated with antenatal or postpartum bleeding, the latter secondary to poor separation.

The presence of one or more small accessory lobes that develop in the membrane at a distance from the main placenta is referred to as ''succenturiate lobe.'' This can be detected sonographically and is clinically important, because retained accessory lobes can be associated with postpartum hemorrhage and infection. Succenturiate lobes are also associated with an increased incidence of velamentous insertion of the umbilical cord and vasa previa.

Bipartite placenta is a placenta that is separated in two and the lobes originate from the anterior and posterior wall of the uterus. The cord can be inserted between the two lobes. Unlike the bipartite placenta, placenta bilobate refers to a placenta where the cord inserts into either lobe but not in the chorionic ridge.

Circumvallate placenta occurs when the membranes insert away from the placental edge toward the center, but a thick chorioamniotic membrane that forms a ridge characterizes this insertion site. On sonographic evaluation, suspicion of a circumvallate placenta occurs if an irregular placental edge, uplifted margin, or placental shelf is seen. Although complete circumvallate placenta has been associated with adverse perinatal outcome, the accuracy of prenatal sonographic diagnosis remains low [45].

Abnormalities of placental location

Establishing location of the placenta in relationship to the internal cervical os is an integral part of every ultrasound evaluation. Placenta previa complicates 1 in 250 to 300 pregnancies. The concept of placental migration was introduced by King [46] and may explain why the incidence of placenta previa is gestational age–dependent. The incidence may be as high as 25% in evaluations performed at 18 weeks gestation. The resolution of a placenta previa seen early in gestation may be caused by growth of the lower uterine segment. A placenta that covers the internal os completely during the midtrimester is more likely to remain a complete placenta previa at term (Fig. 20).

Placenta previa is classified as complete or total when the placenta covers the entire internal cervical os, partial when the os is only partially covered by placenta, and marginal when the edge of the placenta is at the margin of the internal os [47]. Risk factors associated with placenta previa include advanced maternal age, previous cesarean section or uterine scar, multiple gestations, and previous elective abortions [47]. The recurrence risk may be as high as 10-fold.

The diagnostic accuracy of transvaginal sonography is superior compared with the transabdominal approach [48]. The evaluation should take place with the maternal bladder filled and again postvoid. Extensive distention of the bladder may cause apposition of the anterior and posterior uterine walls and lead to an erroneous diagnosis of placenta previa.

Abnormal placentation with myometrial invasion can be a life-threatening condition. Placenta accreta is defined as partial or total absence of the decidua

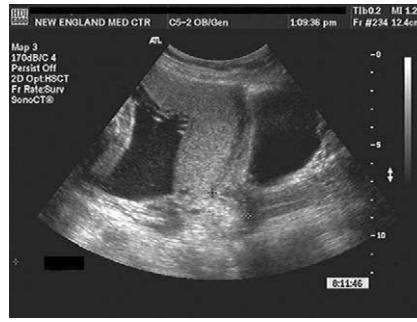

Fig. 20. Complete placenta previa over the cervical os.

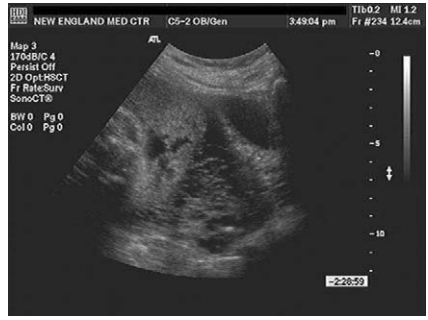

Fig. 21. Suspicion of an accreta with lacunar spaces over the cervical os.

basalis and Nutabuch layer allowing villi to attach to the myometrium. Deeper penetration into the myometrium is referred to as ''placenta increta'' and invasion through the myometrium with potential invasion of adjacent organs is known as placenta percreta [47]. The occurrence of accreta with a placenta previa may be as high as 5%. Risk factors include advanced maternal age and previous uterine surgery. The risk may be as high as 67% in women who have undergone four or more cesarean section deliveries [47].

Antenatal diagnosis caused by increased accuracy of ultrasound and MRI detection has significantly lowered postpartum hemorrhage, maternal morbidity, and mortality. Normally, the retroplacental area is composed of myometrium and uteroplacental vessels that appear hypoechoic and measure about 1 to 2 cm in thickness. Ultrasound criteria for placenta accreta involves careful evaluation of this retroplacental area where there is loss of the normally hypoechoic space and the placental myometrial interface. Markedly dilated spaces called lacunae and increased vascularity may also be present giving this area a Swiss cheese appearance [49]. Transvaginal ultrasound with color Doppler imaging improves visualization and MRI may be helpful to delineate invasion of adjacent organs (Figs. 21 and 22) [50].

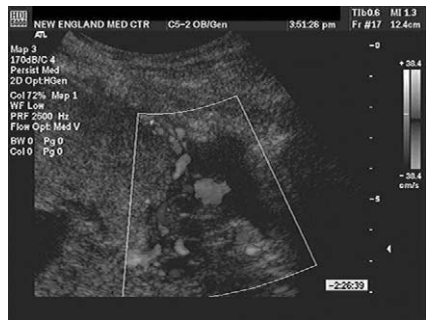

Fig. 22. Color Doppler helps demonstrate large vessels over the cervix.

Placental abruption

Placental abruption complicates approximately 1% of pregnancies and is defined as a premature separation of a normally implanted placenta. Placental abruption may present clinically with abdominal and pelvic pain, vaginal bleeding, or uterine tenderness. Numerous risk factors have been associated with placental abruption, such as maternal hypertension, smoking, cocaine use, trauma, premature rupture of membranes, and uterine anomalies [47].

The sensitivity of ultrasound to visualize a placental abruption is approximately 50% because the appearance may be variable depending on the location of the separation and timing of evaluation [51]. Acutely, the area may appear hyperechoic; however, after 1 to 2 weeks, the area of hemorrhage may become hypoechoic. Although visualization of a thickened retroplacental area may raise suspicion of a retroplacental hemorrhage, uterine contractions, subchorionic cysts, and uterine fibroids may have the same appearance [51]. Fibroids are generally more uniform and round in shape and color Doppler demonstrates increased vascular flow. Subchorionic cysts may be confused with chorioangiomas or placental abruption; however, these cysts are found most often below the chorionic plate and usually have no clinical significance. Separation of the retromembranous area may also be seen. Color Doppler studies may help differentiate these entities because placental abruption lacks vascular activity.

Prognosis depends on several factors including the amount of placental detachment and gestational age. The gravest prognosis is associated with a significant retoplacental hemorrhage involving over 30% to 40% of the placenta and may include fetal growth restriction, oligohydramnios, and preterm delivery (Fig. 23) [52].

Placental masses

Placental tumors are generally benign; however, metastatic lesions from hematogenous spread of conditions, such as metastatic melanoma, may occur. Color flow and pulsed Doppler studies can help differentiate between a vascular and nonvascular lesion.

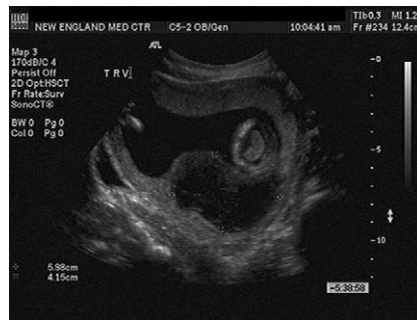

Fig. 23. Retroplacental bleed.

Chorioangioma, also referred to as ‘‘hemangioma,’’ is the most common benign placental tumor [53]. Small chorioangiomas are present in approximately 1% of all examined placentas. Large clinically significant chorioangiomas measuring over 5 cm are rare. These lesions can be associated with fetal morbidity, such as nonimmune hydrops, intrauterine growth restriction, and stillbirth. Preeclampsia, polyhydramnios, and elevated amniotic fluid and maternal serum alpha-fetoprotein have also been reported in association with large chorioangiomas [53].

On ultrasound evaluation, these lesions most commonly protrude from the fetal surface and appear as a solid well-circumscribed mass. Color Doppler evaluation denotes a very vascular lesion, differentiating chorioangiomas from avascular masses, such as fibroids, hematomas, or subchorionic fibrin. Chorioangiomas measuring greater than 5 cm in size warrant fetal evaluation and follow-up to assess for signs of fetal compromise, such as cardiac overload.

Gestational trophoblastic disease

Complete hydatidiform mole is characterized by chorionic villi that are markedly hydropic and swollen and proliferation of the trophoblastic cell resulting in very elevated human chorionic gonadotropin levels. Sonographically, hydatidiform mole has a characteristic appearance. The uterus is large and filled with multicystic hyperechoic or anechoic masses that may correlate to vesicles, the fetus is absent, and there is no amniotic fluid [54]. In the first trimester, vesicles can be detected, although these may not be delineated as easily because the uterine cavity may normally appear hyperechoic (Fig. 24) [55].

The presence of a coexisting fetus is referred to as a ‘‘partial hydatidiform mole.’’ Severe intrauterine growth restriction and fetal anomalies may be present. Karyotype notes triploidy in almost 90% of cases. Sonographic evaluation notes a thickened placenta with multiple cystic spaces (Fig. 25).

Bilateral theca lutein cysts can be seen in up to 50% of cases of gestational trophoblastic disease. These are believed to occur secondary to high circulating

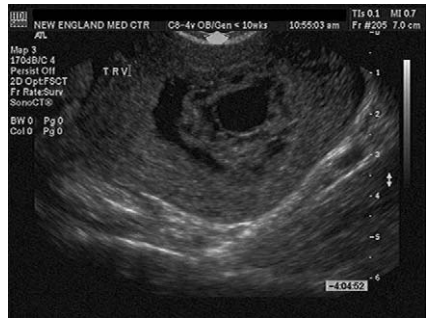

Fig. 24. Ultrasound scan of a complete mole in the first trimester.

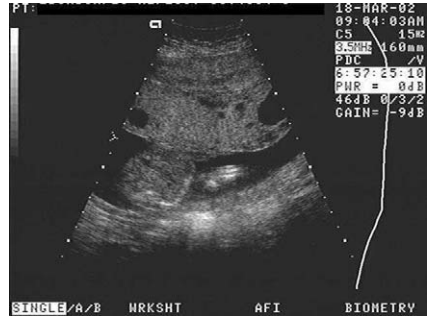

Fig. 25. Ultrasound scan of a partial mole showing multicystic masses in the placenta and presence of a fetus.

levels of stimulating β-human chorionic gonadotropin and appear as large multiloculated simple cysts, which may require months to resolve.

Doppler evaluation

The placental bed spiral arteries undergo progressive physiologic changes throughout gestation. Doppler studies of the umbilical cord are considered an evaluation of the placenta and can assess placental blood flow in pregnancy. The most commonly used measurements include the systolic over diastolic ratio and the resistance index; the latter represents the difference between the peak systolic and end-diastolic shift divided by the peak systolic shift.

In early pregnancy, placental resistance is high and absent end-diastolic velocity normally may be seen between 14 and 18 weeks gestation [56]. A continuous decline in umbilical artery resistance over gestation is normally observed. The progression of increased resistance and loss of end-diastolic velocity or eventual reversal has been associated with adverse pregnancy outcome including intrauterine growth restriction, oligohydramnios, and stillbirth. When used in high-risk pregnancies, Doppler studies can decrease perinatal mortality. Doppler studies should not be used routinely and currently no benefit other than use in the eval-

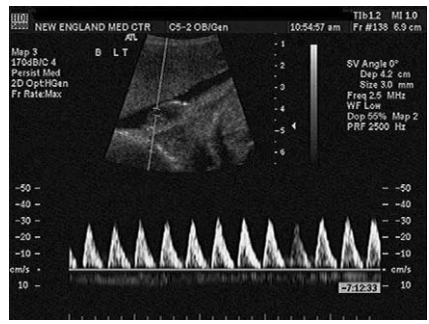

Fig. 26. Color Doppler of umbilical artery showing absent end-diastolic flow.

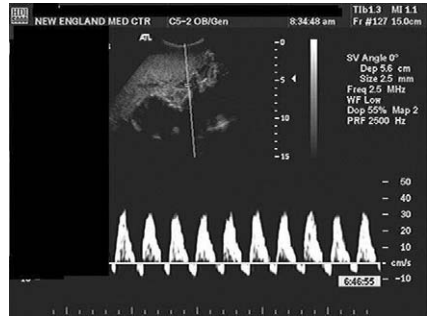

Fig. 27. Color Doppler of the umbilical artery showing reversed end-diastolic flow.

uation of intrauterine growth restriction has been established (Figs. 26 and 27) [57,58].

Summary

Prenatal ultrasound has expanded the ability to assess the umbilical cord, fetal membranes, amniotic fluid volume, and placenta. Evaluation of these structures provides information regarding the intrauterine environment. Umbilical cord abnormalities may be associated with fetal aneuploidy, structural anomalies, and fetal compromise. Estimating the amniotic fluid volume has become an integral part of a sonogram and provides immense information regarding possible fetal anomalies and perinatal outcome. Likewise, placental location or abnormalities may significantly impact obstetric management and prognosis. Early detection of several of these conditions may lead to increased vigilance that may improve perinatal outcome.

References

[1] Brace RA. Progress toward understanding the regulation of amniotic fluid volume: water and solute fluxes in and through the fetal membranes. Placenta 1995;16:1–18.
[2] Brace RA, Wolf EJ. Normal amniotic fluid volume changes throughout pregnancy. Am J Obstet Gynecol 1989;161:382–8.
[3] Dildy GA, Lira N, Moise KJ, Riddle GD, Deter RL. Amniotic fluid volume assessment comparison of ultrasound estimates vs direct measurements with dye-dilution technique in human pregnancy. Am J Obstet Gynecol 1992;167:986–94.
[4] Chamberlain PF, Manning FA, Morrison I, Harman CR, Lange IR. Ultrasound evaluation of the amniotic fluid: the relationship of marginal and decreased amniotic fluid volume to perinatal outcome. Am J Obstet Gynecol 1984;150:245–9.
[5] Phelan JP, Ahn MO, Smith CV, Rutherford SE, Anderson E. Amniotic fluid index measurement in pregnancy. J Reprod Med 1987;32:601–4.
[6] Moore TR, Cayle JE. The amniotic fluid index in normal human pregnancy. Am J Obstet Gynecol 1990;162:1168–72.

[7] Magann EF, Nolan TE, Hess LW, Martin RW, Whitworth NS, Morrison JC. Measurement of the amniotic fluid volume: accuracy of ultrasonographic techniques. Am J Obstet Gynecol 1992; 167:1533–7.
[8] Magann EF, Sanderson M, Martin RW, Chauhan S. The amniotic fluid index, single deepest pocket and 2-diameter pocket in human pregnancy. Am J Obstet Gynecol 2000;182:1581–8.
[9] Shipp TD, Bromely B, Pauker S, Frigoletto FD, Benacerraf BR. Outcome of singleton pregnancies with severe oligohydramnios in the second and third trimester. Ultrasound Obstet Gynecol 1996;7:108–13.
[10] Dyer SN, Burton BK, Nelson LH. Elevated maternal serum alpha-feto-protein levels and oligohydramnios poor prognosis for pregnancy outcome. Am J Obstet Gynecol 1987;157:336–9.
[11] Chauhan S, Sanderson M, Hendrix N, Magann EF, Devoe LD. Perinatal outcome and amniotic fluid index in the antepartum and intrapartum period: a meta-analysis. Am J Obstet Gynecol 1999; 181:1473–8.
[12] Manning FA, Hill LM, Platt LQ. Qualitative amniotic fluid volume determination by ultrasound: antepartum detection of intrauterine growth restriction. Am J Obstet Gynecol 1981;139:254–8.
[13] Rutherford SE, Phelan VP, Smith CV, Jacobs N. The four-quadrant assessment of the amniotic fluid volume: an adjunct to antepartum fetal heart rate testing. Am J Obstet Gynecol 1987;70: 353–6.
[14] Bianco A, Rosen E, Kuczynski M, Tetrokalavili M, Lockwood CJ. Measurement of the amniotic fluid volume with and without color Doppler. J Perinat Med 1999;27:245–9.
[15] Magann EF, Chauhan S, Barrilleaux S, Whitworth NS, McCurley S, Martin JN. Ultrasound estimate of the amniotic fluid volume: color Doppler over diagnosis of oligohydramnios. Obstet Gynecol 2001;98:71–4.
[16] Hill LM, Breckle R, Thomas ML, Froes JK. Polyhydramnios: ultrasonographically detected prevalence and neonatal outcome. Obstet Gynecol 1987;69:21–5.
[17] Dasche JS, McIntire DD, Ramus RM, Santos-Ramos RN, Twickler DM. Hydramnios: anomaly prevalence and sonographic detection. Obstet Gynecol 2002;100:134–9.
[18] Bianchi DW, Crombleholme TM, D'Alton ME, editors. Anencephaly. In: Fetology: diagnosis and management of the fetus as a patient. New York: McGraw-Hill; 2000. p. 63–8.
[19] Panting-Kemp A, Nguyen T, Chang E, Quillan E, Castro L. Idiopathic polyhydramnios and perinatal outcome. Am J Obstet Gynecol 1999;181:1079–82.
[20] Magann EF, Chauhan S, Martin J. Ultrasound assessment of the amniotic fluid volume in diamniotic twins. J Soc Gynecol Invest 1995;2:609–13.
[21] Crombleholme TM, Dirkes K, Whitney TM, Alman B, Garmel S, Connelly RJ. Amniotic band syndrome in the fetal lambs I: Fetoscopic release and morphometric outcomes. J Pediatr Surg 1995;30:974–8.
[22] Mahoney BS, Filly RA, Callen PW, Golbus MS. The amniotic band syndrome: antenatal sonographic diagnosis and potential pitfalls. Am J Obstet Gynecol 1995;152:63–8.
[23] Torpin R. Amniochorionic mesoblastic fibrous strings and amniotic bands: associated constricting fetal anomalies or fetal death. Am J Obstet Gynecol 1965;91:65–75.
[24] Lockwood C, Ghidini A, Romero R, Hobbins J. Amniotic band syndrome: reevaluation of its pathogenesis. Am J Obstet Gynecol 1989;160:1030–3.
[25] Bianchi DW, Crombleholme TM, D'Alton ME, editors. Amniotic band syndrome. In: Fetology: diagnosis and management of the fetus as a patient. New York: McGraw-Hill; 2000. p. 761–70.
[26] Abuhamed AZ, Shaffer W, Mari G, Copel J, Hobbins J, Evans AT. Fetus-placenta-newborn: a single umbilical artery. Does it matter which artery is missing? Am J Obstet Gynecol 1995;173: 728–32.
[27] Nyberg DA, Mahoney BS, Luthy D, Kapur R. Single umbilical artery: prenatal detection of concurrent anomalies. J Ultrasound Med 1991;10:247–53.
[28] Sherer DM, Anyaegbunam A. Prenatal ultrasonographic morphologic assessment of the umbilical cord: a review. Part I. Obstet Gynecol Surv 1997;52:506–14.
[29] Strong TH, Finberg JH, Mattox JH. Antepartum diagnosis of noncoiled umbilical blood vessels. Am J Obstet Gynecol 1994;170:1729–31.

[30] Strong Jr TH, Elliott JB, Radin TG. Noncoiled umbilical blood vessels: a new marker for fetus at risk. Obstet Gynecol 1993;81:409–11.
[31] Sherer DM, Anyaegbunam A. Prenatal ultrasonographic morphologic assessment of the umbilical cord: a review. Part II. Obstet Gynecol Surv 1997;52:515–23.
[32] Jouppill P, Kirkinen P. Ultrasound diagnosis of nuchal encirclement by the umbilical cord: a case and methodological report. J Clin Ultrasound 1982;10:59–62.
[33] Jauniaux E, Ramsay B, Peellaerts C, Scholler Y. Perinatal features of pregnancies complicated by nuchal cord. Am J Perinatol 1995;12:255–8.
[34] Hanaoka U, Yanagihara T, Tanaka H, Hata T. Comparison of three-dimensional, two-dimensional and color Doppler in predicting the presence of a nuchal cord at birth. Ultrasound Obstet Gynecol 2002;19:471–5.
[35] Nyberg DA, Filly RA, Globus MS, Stephens JD. Entanglement of the umbilical cord: a sign of monoamniotic twins. J Ultrasound Med 1984;3:29–32.
[36] Fung TY, Lau TK. Poor perinatal outcome associated with vasa previa. Is it preventable? A report of three cases and review of the literature. Ultrasound Obstet Gynecol 1998;12:430–3.
[37] Nomiyama M, Toyota Y, Kawano H. Antenatal diagnosis of velamentous cord insertion and vasa previa with color Doppler imaging. Ultrasound Obstet Gynecol 1998;12:426–9.
[38] Craven C, Ward K. Placental causes of fetal malformations. Clin Obstet Gynecol 1996;39: 588–606.
[39] Ross JA, Jurlovic D, Zosmer N, Jauniaux E, Hacket E, Nicolaides KH. Umbilical cord cysts early in pregnancy. Obstet Gynecol 1997;89:442–5.
[40] Mahoney BS, McGahan JP, Nyberg DA, Reisner DP. Varix of the fetal intra-abdominal umbilical vein: comparison with normal. J Ultrasound Med 1992;11:73–6.
[41] Kuhlmann RS, Warsof S. Ultrasound of the placenta. Clin Obstet Gynecol 1996;39:519–34.
[42] Wolf H, Oosting H, Treffers PE. Second trimester placental volume measurement by ultrasound: prediction of fetal outcome. Am J Obstet Gynecol 1989;160:121–6.
[43] Spirt BA, Gordon LP. The placenta as an indicator of fetal maturity: fact and fancy. Semin Ultrasound CT MR 1984;5:290–7.
[44] Pinette MG, Loftus-Brault K, Nard DA. Maternal smoking and accelerated placental maturation. Obstet Gynecol 1989;73:379–82.
[45] Harris RD, Wells WA, Black WC, Chertoff JT, Poplack SP, Sargent SK, et al. Accuracy of prenatal sonography for detection of circumvallate placenta. AJR Am J Roentgenol 1997;168: 1603–8.
[46] King DL. Placenta migration demonstrated by ultrasonography: a hypothesis of dynamic placentation. Radiology 1973;109:167–70.
[47] Cunningham FG, MacDonald PC, Gant NF, Leveno KJ, Gilstrap LC, Hankins GV, et al. Obstetrical hemorrhage. In: Williams obstetrics. 21st edition. New York: Appleton and Lange; 2001. p. 619–69.
[48] Farine D, Peisner DB, Timor-Trisch IE. Placenta previa: is the traditional diagnostic approach satisfactory? J Clin Ultrasound 1990;18:328–30.
[49] Cox SM, Carpenter RJ, Cotton DB. Placenta percreta: ultrasound diagnosis and conservative surgical management. Obstet Gynecol 1988;71:454–6.
[50] Levine D, Hulka C, Ludmir J. Placenta accreta: evaluation with color Doppler, power color Doppler and MR imaging. Radiology 1997;205:773–6.
[51] Nyberg DA, Cyr DR, Mack LA, Wilson DA, Schuman WP. Sonographic spectrum of placental abruption. AJR Am J Roentgenol 1987;148:161–4.
[52] Nyberg DA, Mack LA, Benedetti TJ, Cyr DR, Schuman WP. Placental abruption and placental hemorrhage: correlation of sonographic findings with fetal outcome. Radiology 1987;164: 357–61.
[53] Shalev E. Prenatal diagnosis of placental hemangioma and clinical implications; a case report. Int J Gynecol Obstet 1984;22:291–3.
[54] Wagner BJ, Woodward PJ, Dickey GE. From the archives of the AFIP. Gestational trophoblastic disease: radiologic-pathologic correlation. Radiographics 1996;16:131–48.

[55] Lazarus E, Hulka C, Siewert B, Levine D. Sonographic appearance of early complete molar pregnancies. J Ultrasound Med 1999;18:589–94.
[56] Harman CR, Baschat AA. Comprehensive assessment of fetal well being: which Doppler tests should be performed? Curr Opin Obstet Gynecol 2003;15:147–57.
[57] American College of Obstetrics and Gynecology. Intrauterine growth restriction. Washington DC: The American College of Obstetricians and Gynecologists; Practice Bulletin. No. 12. January 2000.
[58] American College of Obstetrics and Gynecology. Antepartum fetal surveillance. Washington DC: The American College of Obstetricians and Gynecologists; Practice Bulletin No. 9. October 1999.