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Sonographic Assessment of the Umbilical Cord

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INTRODUCTION

For centuries there has been fascination with umbilical cord. The cord itself, as well as the remnant of the umbilical cord, a navel (umbilicus) on the adult abdomen, had ascribed mystical powers in ancient[1,2] and some present cultures.[3] It represents the "life source", or the "entry" and "exit" point of human will and source of energy that makes connection to the surrounding world. Regardless of umbilical cord mystical powers, it was always obvious that it provides the life support to the fetus, and therefore, represents the major fetomaternal unit. All building blocks required by the developing fetus are exchanged with waste materials through this blood flow highway. As such, any morphologic umbilical cord abnormalities will affect blood flow and modulate function of the end units that the same connects-placenta and fetus. Interestingly, despite of the paramount importance of the umbilical cord, it is a relatively simple structure that is usually composed of two arteries and one vein. These blood vessels are surrounded with Wharton's jelly, a gelatinous stroma, and covered by a single layer of amnion.[4] Such structure provides flexibility, mobility and strength to resist compression, whilst at the same time allows the fetus to move freely.

The umbilical cord can be easily demonstrated and assessed by conventional real-time ultrasound.[5] In addition to morphologic cord's characteristics that would be described by the number of vessels, the umbilical blood flow patterns can be analyzed by color (power) and pulsed Doppler ultrasound that relate to its functionality.[6] It can provide more clinically useful information about the placental state, as well as impeding fetal jeopardy. Though we recognize the umbilical cord importance, unfortunately, we usually assess only one of the many umbilical cord features—the number of the cord vessels. This is routine part of the second trimester fetal anatomic survey, and any other umbilical abnormalities, e.g. cystic changes, or abnormal cord thickness and/or twisting are usually described as an incidental finding. In contrast, if specialized ultrasound examination is utilized, in the subgroup of fetuses with intrauterine growth restriction for example, the analysis of the umbilical cord blood flow has been found to be clinically useful. In these patients, the pulsed Doppler analysis of the umbilical cord artery and its resistance to blood flow is indirectly used to assess placental function as previously mentioned.[7] The rest of the umbilical cord's features are considered as investigational and largely ignored, though some evidence exist that cord thickness and amount of Wharton's jelly, excessiveness or lack of cord coiling, presence of functional Hyrtle anastomosis, and umbilical vein blood flow patterns, could be useful in evaluation of various clinically adverse ante- and perinatal events.

UMBILICAL CORD DEVELOPMENT

From the development of the embryonic pole, and thereafter an embryo, the umbilical cord is formed from the stalk of the yolk sac that becomes covered with single layer of amniotic epithelium due to expansion of the amniotic sac.[4] At these early stages of development, the primitive umbilical cord contains allantois with allantoic vessels that will form definitive umbilical vessels, and vitaline duct with omphalomesenteric vessels. The former allantoic vessels will provide two umbilical arteries and two veins, and a week later, umbilical veins will form the venous network with omphalomesenteric veins in the developing liver thereby establishing the umbilical-portal venous connection. Interestingly, by the eight week of gestation, the right umbilical vein commonly regresses, forming the commonly known umbilical cord with the one vein and two arteries. The remaining vein enlarges to accommodate the increasing blood flow. The left umbilical vein enters the left portal vein directly and with development of the ductus venosus, a larger portion of blood volume enters directly into the systemic venous system bypassing the liver venous network. Approximately at the same time, umbilical cord's helical pattern (coiling) starts to develop. It is unclear how this process is regulated, though a possibility that cord twisting occurs due to discordant vessels' growth

simultaneously with the regression of the one umbilical vein, is most likely.

Growth of the umbilical cord parallels growth of the fetus until 28 weeks, when the umbilical cord attains its final mean length of 50 to 60 cm, with the mean circumference of 3.6 cm.[8] The umbilical arteries convey deoxygenated blood from the fetus to the placenta. Intrabdominal portion of the umbilical arteries run along-side of the fetal bladder originating from the internal iliac vessels. The intra-abdominal portions of the umbilical arteries regress and degenerate postnatally into the lateral ligaments of the bladder, while the umbilical vein becomes the round ligament of the liver.

UMBILICAL CORD ANATOMY

There are several major umbilical cord morphologic characteristics amenable to ultrasound evaluation. The most common description of the cord is by the total number of umbilical cord vessels. In addition to number of cord vessels, the umbilical cord thickness and the amount of Wharton's jelly, coiling (helical pattern or twisting), location of placental cord insertion, presence of the Hyrtle anastomosis, and the blood flow characteristics of the cord vessels (vein and both umbilical arteries) can be assessed.

UMBILICAL CORD LENGTH

The umbilical cord length is one of the major cord morphological features that cannot be accurately assessed antenataly via conventional ultrasound. An abnormal umbilical cord length, either excessive or short, is a known risk factor for adverse perinatal outcome. A short umbilical cords, less than 35 cm in length, were associated with congenital anomalies, reduced fetal activity, interference with heart-rate patterns in labor, restriction of fetal descent, and cord rupture.[4] Similarly, long cords in excess equal and above 70 cm in length are described in association with fetal entanglement, true knots, torsion and prolapse.[9] Therefore, it would be very important to develop means of ultrasound assessment of cord length. Up to date, there are few studies that measured umbilical cord length in first and second trimester of pregnancy.[10,11] Collins measured umbilical cord length in 30 fetuses at 28 weeks gestation, and was able to depict one case with abnormally long cord, more than 50 cm in length. In contrast, Hill et al, assessed umbilical cord length between 6 to 11 weeks gestation, and observed linear relationship between cord length and menstrual age in the normal group. Interestingly, in 60% of fetuses (9 out 15) who had intrauterine fetal demised, cord length was more than 2SD below the expected value for menstrual age in normal fetuses. Currently, measurements of umbilical cord length except as a subjective assessment solely, are not widely applied in clinical obstetrics.

However, there we hope that with utilization of 3D ultrasound, more objective means of cord length measurements will be produced. At the present time, we are in progress with the study that assess umbilical cord length in the first trimester. With the help of 3D ultrasound and postprocessing 3D features, we adjust the image of the cord by rotating X, Y or Z plane to obtain full umbilical cord for measurement (Figs 1 and 2). Our preliminary data demonstrate large variation in a cord length, though our numbers are still limited to produce a nomogram of the umbilical cord length between 9 to 14 weeks gestation.

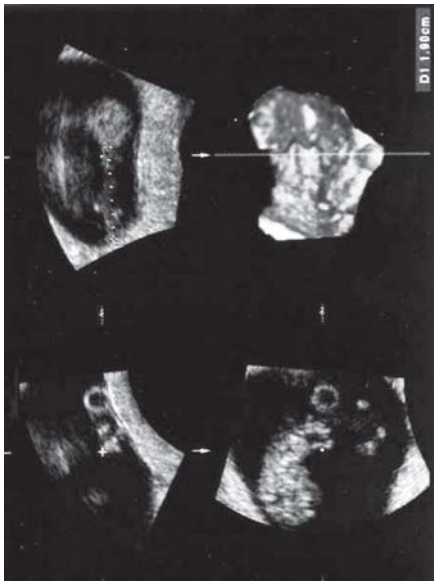


Fig. 1: First trimester fetus assessed by 3D ultrasound. Note position of the fetus and umbilical cord in X, Y, Z planes to obtain full umbilical cord length for measurement

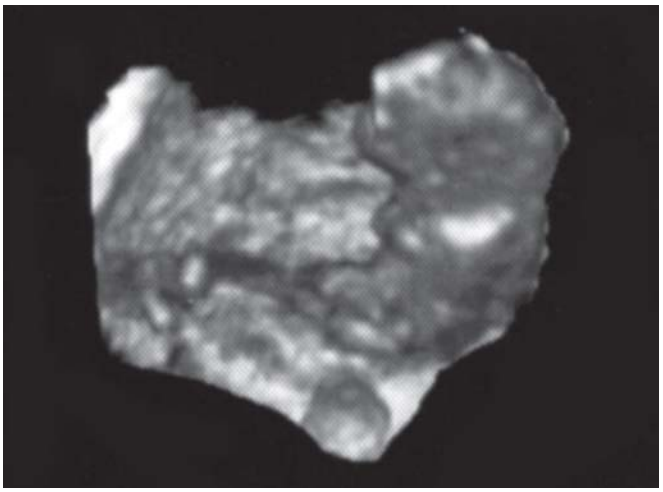


Fig. 2: 3D image of the fetus with complete cord length

UMBILICAL CORD THICKNESS AND WHARTON'S JELLY

The umbilical cord thickness depends on vessels' luminal diameters and amount of Wharton's jelly. In addition to assessment of vessels luminal diameters, an amount of Wharton's jelly can be separately evaluated by subtracting vessels' areas from the total cord area on transverse section of the umbilical cord. An antenatal nomograms of the umbilical cord thickness from 10 to 42 weeks[11-14] and Wharton's jelly area between 15 to 42 weeks of gestational age have been described.[15,16] A generated nomograms demonstrated an increase in umbilical cord thickness as a function of gestational age up to 34-36 weeks of gestation, followed by a reduction of umbilical cord size.[12,15] These morphologic cord characteristics have been associated with adverse ante- and perinatal outcomes. Thin umbilical cords were found to accompany unexplained fetal death, whereas umbilical cord enlargement was observed in association with fetal hydrops, rhesus sensitization or twin-twin transfusion.

The umbilical cord thickness, or the size of cord's cross-sectional area, was found to correlate with fetal biometry.[15,16] It was observed that umbilical cord width cross-sectional area below the 10th percentile for gestational age, categorized as 'lean' umbilical cord, considerably increases the risk of having a small for gestational age (SGA) fetus at delivery, fetal distress in labor, and operative delivery.[17] Similar observations were found in patients with an early onset pre-eclampsia where the umbilical cords were found to be lean with reduced Wharton's jelly amount and smaller umbilical vein area.[18] In contrast, the umbilical cord was significantly larger in fetuses of mothers with gestational diabetes compared to the normal population, and the main increase in cord's width was attributed to an increase in the Wharton jelly content.[12]

Regardless which nomogram is used, it is not yet clear does the umbilical cord thickness follow the fetal growth pattern (as any other fetal biometric feature), or rather it is the predecessor of the altered fetal growth (Fig. 3). In the recent study, it was demonstrated that umbilical cord thickness correlates with estimated fetal weight but does not predict fetal growth deficiency recognized as small for gestational age at birth. However, the major limitation of this study was lack of Wharton's jelly area assessment as possible independent predictor of altered fetal growth as noted by Raio and coworkers. Nevertheless, due to simplicity of the umbilical cord diameter measurement *versus* cord cross-sectional area, it is likely that umbilical cord width/thickness, measured in close proximity to the fetus (e.g. 0.5 cm away from the abdominal cord insertion),[14] would be easier to perform likely with similar results to complete cord cross-sectional area assessment.

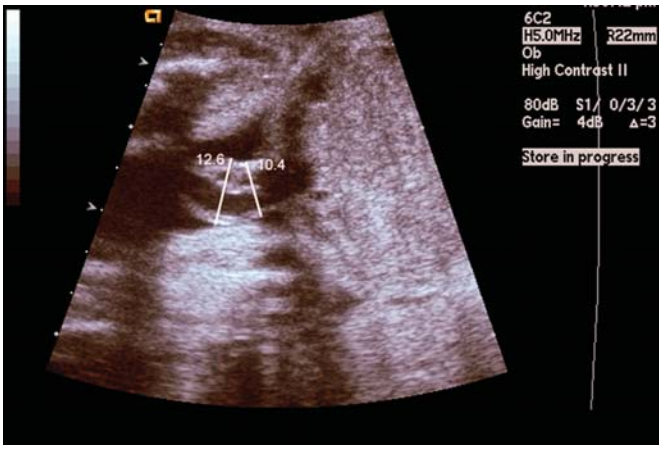


Fig. 3: Umbilical cord insertion into the fetal abdominal wall. Note different measurements of the umbilical cord thickness pending on the distance from the cord insertion. Measurement should be taken at the same distance, e.g. 5 mm from its attachment to the fetal abdomen to provide uniform measurements

An association between aneuploidy in the first trimester of pregnancy and umbilical cord thickness has been recently described.[19] It was noted that aneuploid fetuses have a thicker umbilical cords that is evident in first[19] and second trimester of pregnancy.[20] However, this finding was not confirmed by others.[21] Abnormal umbilical cords' thickening was attributed to the swelling of Wharton's jelly. Several studies observed an alteration of the extracellular matrix in fetuses affected by trisomies 13, 18 and 21.[22,23] These alterations were related to a different expression of structural proteins, mainly polysaccharides and proteglycans of the extracellular matrix, which likely resulted in abnormal fluid accumulations. Someone may speculate that similar pathophysiologic processes could be the possible cause of an umbilical cord swelling as well as increased nuchal translucency observed in aneuploid fetuses.[24] Regardless of the mechanism, it appears that increased umbilical cord thickness is a transient feature. The majority of aneuploid fetuses with thick umbilical cords were observed between 15 to 18 gestational weeks with a trend toward smaller number of thicker umbilical cords at the later gestational age.[20] In addition, the majority of the fetuses (66.6%) were found to have abnormal first trimester genetic screening test. These observations suggested similar pathophysiologic process between nuchal translucency and umbilical cord swelling in aneuploid fetuses. Therefore, the natural tendency towards abnormal umbilical cord thickness resolution could be expected at a later gestation. A stratification of results by abnormal karyotypes showed that 57.8% and 50% of fetuses with trisomy 21 and trisomy 18, respectively, had thick umbilical cords.

In summary, the assessment of umbilical cord thickness, amount of the Wharton's jelly, and or vessels' diameters, mainly

umbilical vein, could be used to recognize a subgroup of patients who are at higher risk of intrauterine growth patter alteration. Leaner, or thin umbilical cord could represent a risk for intrauterine fetal growth deficiency, while thick umbilical cords observed in fetuses with maternal diabetes mellitus, could be associated with excessive fetal weight gain and possible macrosomia development. In contrast, the value of umbilical cord thickness assessment in the first and second trimester of pregnancy, and its value as prognosticator of fetal aneuploidy, are less clear. Although an association with thicker umbilical cords and fetal aneuploidy was observed, the most recent study clearly refuted this association. In a total of 10,000 patients screened in the first trimester of pregnancy for nuchal translucency, no association between umbilical cord thickness and higher prevalence of fetal aneuploidy was found. Therefore, although initially it appeared that aneuploid fetuses demonstrate thicker umbilical cords than euploid fetuses, the importance of the cord thickness as a prognosticator of an abnormal karyotype has not been confirmed in large clinical study.

NUMBER OF UMBILICAL CORD VESSELS

The umbilical cord derives from the yolk sac stalk. At 6 weeks of gestational age, two umbilical arteries that carry blood from fetus to placenta, and two paired umbilical veins that carry blood to fetus from developing placenta, are observed. By the 8th week, the right umbilical veins regress, while the left umbilical vein enlarges to accommodate the increasing flow and enters the left portal vein directly. In contrast, two umbilical arteries will continue to persist till delivery. The disappearance of the right umbilical vein is likely related to the development of ductus venosus in its role as a vascular shunt between the systemic blood flow (vena cava inferior) and hepatic portal system.

The most common umbilical cord anomaly observed by ultrasound is absence of one artery, a condition called a *single umbilical artery (SUA)*. Congenitally absent umbilical artery is described as agenesis, whilst small and poorly developed artery, therefore easily missed by ultrasound, is named as a hypoplastic. It mainly forms when one of the umbilical arteries undergoes atresia, aplasia, or agenesis. The incidence of a SUA ranges between 0.2 to 1.6% among euploid and 9 to 11% among aneuploid fetuses.[25,26] An SUA is commonly associated with fetal malformations, or it can be observed as a single ultrasound finding, named an *isolated SUA* (Fig. 4). Although the umbilical cord can be easily visualized on prenatal sonogram, there is a discrepancy between the theoretic ultrasound accuracy and actual reliability to detect SUA reflected in the range of positive predictive values of 65 to 94%.[25,27] Due to lower than expected ultrasound detection of SUA, there is a discrepancy in number

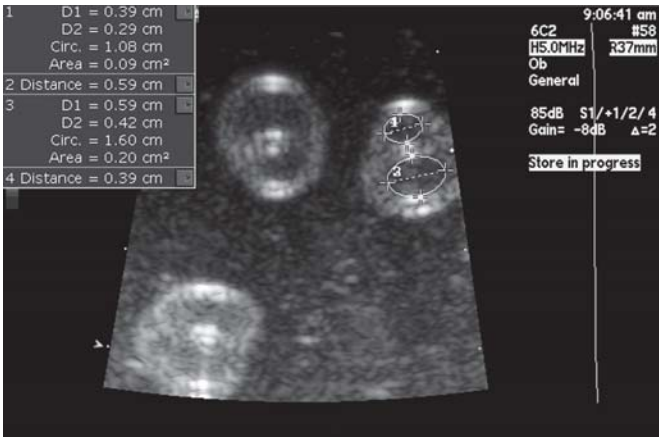


Fig. 4: Single umbilical cord in transverse section. Areas of the vein and artery are measured to obtain vein to artery ratio. Ration above 2.0 could be associated with suboptimal blood flow through the umbilical cord and alter fetal growth potentioal

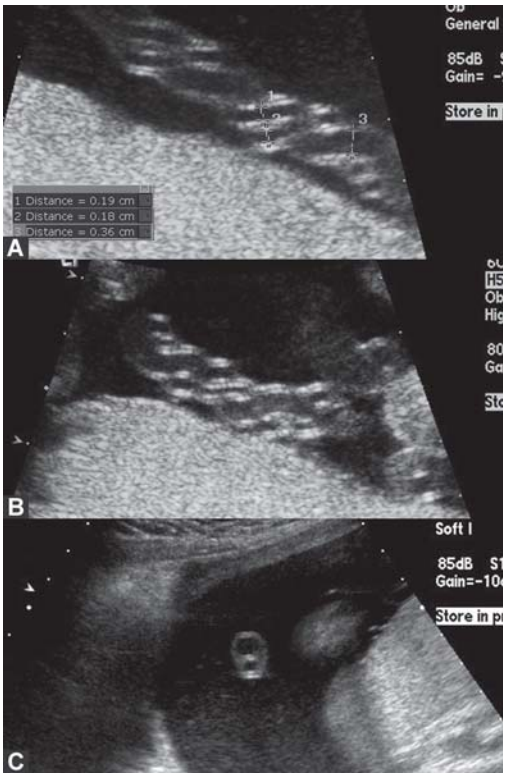
of neonates born with an SUA and associated anomaly diagnosed postnatally compared to those in the antenatal period. The proportion of identified fetuses with associated malformations diagnosed prenatally would rise postnataly from 26 and 43 to 43% and 50%, respectively.[27,28] In addition to associated fetal anomalies with an SUA, an intrauterine fetal growth restriction (IUGR) was observed more frequently in aneuploid than euploid fetuses, 28% *vs* 15-20%, respectively.[29] In those euploid fetuses with an isolated single umbilical artery (iSUA), IUGR was also observed in 15 to 18% of cases.[27,28] Concomitantly, 25-29% of the neonates with an iSUA would have a birth weight less than 2500 gm.[29,30] The difference in birth weights was even more pronounced in multifetal gestation in which one of the twins would have a SUA. Those twins would be on average 500 gm smaller than the cotwin with a three-vessel cord, at average 1730 gm *vs* 2280 gm, respectively.[29] From these reports, it appeared that fetuses with an iSUA finding are at increased risk for IUGR, as well as small-for-gestational age (SGA) at birth. Finally, an iSUA was related to 16.2% of neonates that were found to have some form of renal anomaly, unrecognized prenatally by ultrasound, though in 54.5% of these neonates, the anomalies were minor or self limiting.[31]

The presence of SUA requires meticulous fetal anatomic survey to exclude any additional fetal anomalies. Fetal karyotyping should be considered when additional fetal malformations are observed. If an isolated SUA, serial sonograms for fetal growth and close obstetric follow-up were recommended.[27] However, an association between an isolated SUA and fetal growth deficiency has not been consistently observed.[32] In this study the prevalence of intrauterine fetal growth deficiency was similar between the fetuses having

isolated SUA and control group, while serial ultrasound detected growth deficiency in 50% of cases only. Therefore, the need for serial ultrasound for fetal growth assessment in fetuses with an isolated SUA was questioned. Regardless of the perinatal outcome, fetuses with SUA have to be evaluated postnataly for possible subtle genitourinary anomaly that is associated up to 16% of cases, as mentioned before. Interestingly enough, in contrast to the right umbilical vein that naturally regress, if SUA is encountered, it is more commonly left umbilical artery that is absent. The absence of left or right umbilical artery was found to have no correlation with higher prevalence of ipsilateral renal anomalies.

VESSELS LUMINAL DIAMETERS AND DISCORDANT IN SIZE UMBILICAL ARTERIES

After the placenta cord insertion, umbilical arteries separate and supply one placental lobe. Each of them divides into the several end-placental arteries that end in the center of the corresponding placental cotyledons. Usually, both umbilical arteries are of similar diameter, and sum of them roughly correlates with the vein diameter. However, discordance in the arteries' diameters was associated with placental abnormalities, variation of the umbilical cord insertion and some clinical conditions, such as gestational diabetes.[33] The associated placental pathology is similar to those when single umbilical artery is found, which support the hypothesis that smaller umbilical artery is a variant of hypoplastic vessel that in the worse form represents single umbilical artery (Figs 5A to C).



Figs 5A to C: A transverse section of the umbilical cord reveals number of the cord vessels. Normal umbilical cord contains one vein and two arteries with similar vessels' diameters (A) or discordant umbilical arteries in size (B) An absence of one of the umbilical cord arteries is named a "single umbilical artery" cord (C)

The presence of discordant umbilical arteries is not only evident by different diameters, but also as a sign of different umbilical artery blood flow indices.[6,34] Smaller artery has higher resistance to blood flow than larger one.[33] These findings support the hypothesis that discrepancies in either size or Doppler flow velocities between the two umbilical arteries represent the expression of placental macroscopic or microscopic abnormalities in the territories supplied by the smaller artery.[35] Nevertheless, a small interarterial vessel, named Hyrtl anastomosis, is usually present within 3 cm from the placental cord insertion that connects both arteries and acts as an arterio-arteriousus vascular shunt between these vessels and, therefore, both placental lobes. The Hyrtl anastomosis is found in 95% of all palcentae.[4] and its likely role as a pressure-equalizing system between the placental lobes may be particularly important during uterine contractions when the blood pressure and resistance in the corresponding portion of the intervillous space and cotyledons may.[36] The maturation of Hyrtl anastomosis was noted with the advancing gestation. A discordant blood flow patterns that were observed in more than 20% of the cases would decrease to less than 10% at term, suggesting that "equalizing system" changes its function during the gestation.[6,34] It was also noted that Hyrtl anastomosis was found frequently absent in those placentas where marginal or velamentous placental cord insertion is observed. Therefore, it appears that the presence and functionality of the Hyrtl anastomosis is likely related to the placental morphologic characteristics because no difference in anastomosis patterns or anastomosis diameter was observed between AGA and SGA fetuses.[33] Nevertheless, the true importance of Hyrtl anastomosis and discordant umbilical arteries could be found when umbilical artery pulsed Doppler analysis is employed to help in management of affected intrauterine pregnancies. If the pulsed Doppler signal is not taken from both arteries, but rather from the smaller in diameter artery only, a higher resistance index could be erroneously used as a representative one, when in reality it could present a false positive result.[6]

UMBILICAL CORD COILING

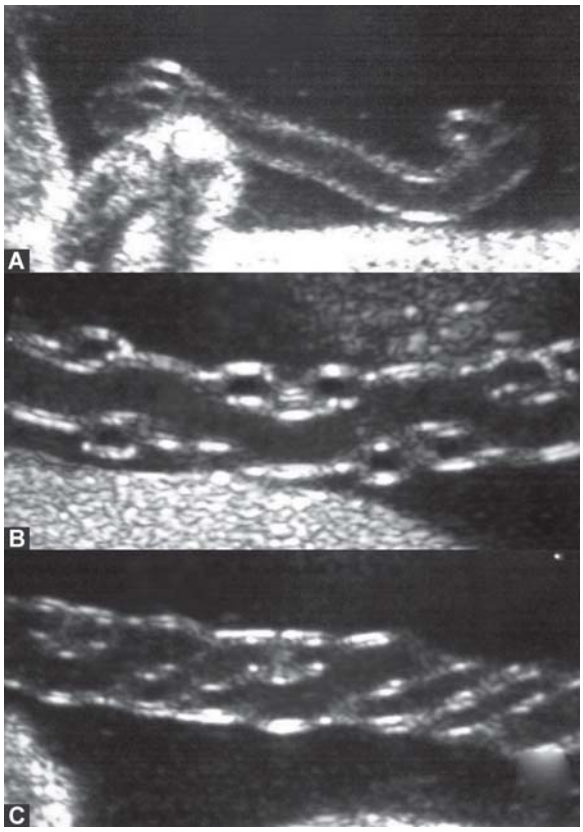
The umbilical cord supposes to be prone to compression, tension or torsion with subsequent interruption of blood flow. It is believed that coiling provides a protective effect to these forces, therefore, securing interrupted blood supply to the fetus. The true etiology of umbilical coiling is unclear, but it is thought to result from fetal movement as well as unequal vascular growth.[37,38] It appears that umbilical coiling pattern is established in the first trimester, although the presence of a mixed coiling pattern and even reversal of coiling direction in third trimester has been demonstrated.[39]

The normal umbilical cord coiling is approximately one coil per 5 cm of umbilical cord length, or 0.20-0.24 coils per cm.[40-43] The latter number represent umbilical coiling quantified via umbilical coiling index (UCI).[44] The UCI is calculated by dividing the total number of coils by the total length of the cord immediately after delivery.[42] The majority of the published studies define hypocoiled (undercoiled) or hypercoiled (overcoiled) umbilical cords as below the 10th and above the 90th percentile, respectively.[41,43,44] In one of the largest studies of 1329 umbilical cords, a total of 13% hypocoiled and 21% of hypercoiled umbilical cords were found.[40] The presence of hypo- and hypercoiled cords was associated with fetal demise (21%, 37%, respectively), fetal intolerance of labor (15%, 14%, respectively), and intrauterine growth restriction (29% and 10% respectively).[40] These findings were confirmed by others,[41-43] and adverse fetal outcomes were attributed to abnormal coiling those likely predisposed umbilical cord vessels to thrombosis and/or constriction.[40]

In an attempt to prognosticate adverse fetal outcomes, an ultrasound evaluation of the umbilical cord coiling demonstrated a significant correlation between antenatal UCI (aUCI) and postnatal UCI measurements, but these studies were limited to the third trimester or the immediate postpartum period.[43-45] However, an attempt to establish the correlation between aUCI obtained in the second trimester and UCI at delivery demonstrated less compelling results with a sensitivity of 78.9% and 25.4% to predict hypo- and hypercoiled umbilical cords, respectively.[46] Discordance between aUCI and UCI may be attributed to the presence of "mixed" coiling patterns or possibly to the evolution of UCI at latter gestation. Nevertheless, a significant correlation between the antenatal UCI and true UCI at term is clearly present. Therefore, an aUCI, determined as a reciprocal value of the distance between the two umbilical coils, can be used predict true UCI at birth.[46] If translated into the distance between coils, the distance obtained by ultrasound at approximately 20 weeks of gestation would be one half of the distance at term. This observation is likely related to the length of the cord and numbers of coils. If a certain number of coils is present in the 50-60 cm long umbilical cord at a term birth, it is believed that the same number of coils would be present at 20 weeks of gestation when a cord length would likely be one half (approximately 30 cm) of the cord length at term. This simple comparison suggests a rough correlation of aUCI *vs* UCI with a ratio 2 to 1.

It is important to stress that the distance between the pair of coils has to be measured in the same manner - calipers should be placed along the ipsilateral side of the cord without crossing or measuring the distance in the middle of the cord. Even more, if the cord is significantly curved, we recommend tracing a distance along the outer edge of the cord, similar to the measurement of cervical length in curved cervices. This would provide a means to standardize the antenatal ultrasound measurement of the umbilical cord coiling (Figs 6A to C and 7).

Antenatal assessment of the UCI at 20 weeks' gestation ultrasound could be used to predict perinatal adverse outcomes. In recent study, we observed a statistically significant association between an abnormal coiling pattern and higher



Figs 6A to C: An ultrasound demonstration of umbilical cord coiling patterns. The umbilical cord has different coiling patterns based on the frequency of the umbilical twists; from hypocoiled or undercoiled (A) normocoiled (B) to hypercoiled or overcoiled umbilical cords (C)

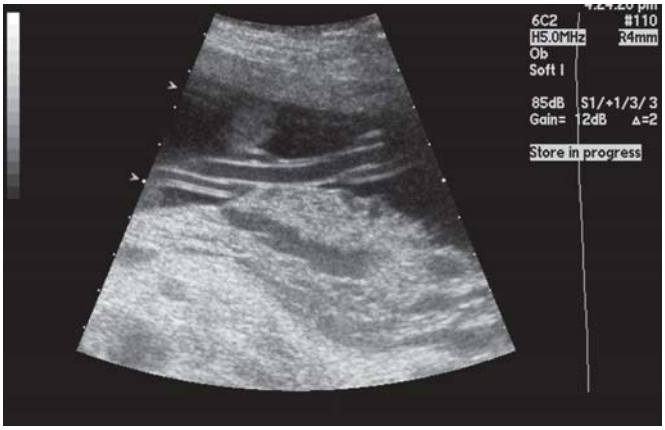


Fig. 7: Umbilical cord hypocoiling associated with placental lakes. Abnormal coiling and presence of multiple placental lakes should promt more careful observation of the fetal growth pattern due to possible suboptimal placental function related to these anatomical abberations

prevalence of SGA neonates at birth, and nonreassuring fetal status in labor.[47] These results were in agreement with previously published data that noted high predictive value to detect SGA fetuses via aUCI assessed at 15 weeks of gestation.[45] This study demonstrated sensitivity rate of 79% and specificity of 86%, when compared to other modalities of the cord evaluation such as cord thickness and Doppler studies in prediction of SGA fetuses. The authors used a cut of value of 0.29 to discriminate hypocoiled from normocoiled umbilical cords, and therefore, linked hypocoiling with higher prevalence of SGA infants at birth. In contrast to their results, we observed a higher prevalence of SGA neonates at similar rates on both ends of the coiling spectrum.[47] A trend towards a higher prevalence of interventional deliveries and meconium stained amniotic fluid in labor were also noted in fetuses with hypo- and hypercoiled umbilical cords.[47] These findings were not in agreement with previously published reports of a statistically higher prevalence of meconium staining, interventional delivery, intrapartum fetal heart-rate disturbances, in association with abnormal coiling[42,43] or hypocoiled cords only,[29] as well as fetal intolerance of labor, growth restriction[40] or emergency Cesarean deliveries.[29] Not all reports found an association between umbilical coiling patterns and adverse pregnancy outcome. No significant correlations of the UCI with maternal age, parity, gestational age at delivery, mode of delivery, fetal gender or birth weight was found in a study of 122 umbilical cords.[48] It is possible that the small number of subjects in this study[33] precluded observation of adverse pregnancy outcomes in those patients with abnormal umbilical cord coiling that was reported elsewhere.[39,42-44] In regard of neonatal Apgars' scores at birth, a higher number of low scores (≤ 6), though not statistically significant, in association with hypercoiled umbilical cords were reported.[42,47]

In majority of the umbilical cords a twisting of arteries over umbilical vein is observed. Interestingly enough, a small proportion of umbilical cords (4.2%) with twisting patterns where significant coiling of the vein around the straight or minimally coiled arteries, can be noted.[48] One half of these cords were hypercoiled, and the other half also had high aUCI index. Due to the hypercoiling pattern of these cords, one could expect a higher incidence of adverse events in labor than was observed in this group. However, the incidence of adverse events was similar to the group of patients with a normal coiling pattern, which implicates the possibility of a mechanical protective effect against blood flow interruption when the vein is coiled around the arteries.[48] In this manner, the vein is not susceptible to collapse when the arteries are in state of spasm due to any reason, compression or hypoxemia.

In summary, regardless of the possible pathophysiology of abnormal umbilical cord coiling and its effect on the pregnancy and the fetus in labor, a presence of hypo- and hypercoiling of the umbilical cord, observed during the fetal ultrasound anatomical evaluation in the second trimester, is associated with adverse perinatal outcomes. It can potentially be used as a predictor of an adverse antenatal and perinatal events in future studies.

OTHER UMBILICAL CORD ABNORMALITIES

Umbilical Vein Aneurysm

The umbilical vein aneurysm, or varix, represents a focal dilatation of the umbilical vessels, umbilical vein or the artery. It is a rare finding and was observed sporadically in fetuses with no adverse perinatal outcomes, though noted in 3.8% of cases that resulted in perinatal death.[49] The etiology of this finding is unknown, though could be related to any condition that increased vessel pressure. The umbilical vein varix is commonly seen at intra-abdominal and extrahepatic portion of the umbilical vein.[50,51] The umbilical vein varix is diagnosed if the vein diameter is greater than 8 mm,[51] or the lumen of the varix measures 50% larger diameter that the intrahepatic portion of the umbilical vein.[52] The significance of the umbilical vein varix is related to the presence of associated fetal abnormal findings, and adverse pregnancy outcome. Few reports associated umbilical vein varix with the fetal chromosomal abnormalities.[52,53] Whereas, others reported adverse pregnancy outcomes, such as fetal hydrops and perinatal death, in these pregnancies.[50,52,53] It is important to note that in majority of fetuses with adverse pregnancy outcomes, associated anomalies were observed. Therefore, the

umbilical vein varix increased the risk of the adverse pregnancy outcome, though if observed as an isolated findings, it had favorable prognosis.[54] At present time, there is controversy weather the presence of the umbilical vein varix warrants a change in obstetrical management. If the umbilical vein varix is observed with associated fetal anomalies, or chromosomal abnormality, obstetrical management will depend on the later findings. In contrast, if the umbilical vein varix is an isolated finding, there is no clear evidence that would support change in obstetrical management. However, some authors believe that close fetal monitoring should be initiated and delivery should be strongly contemplated once the fetal lung maturity is accomplished, or fetal distress is apparent.[55]

Umbilical Cord Lesions

During the assessment of the umbilical cord, a variety of cord lesions are encountered incidentally. These rare masses vary from the pseudocord lesions such as omphalocele or gastoschisis, to true lesions such as urachal, allantoic or omphalomesenteric duct cysts, hemangioma or hematoma, and mucoid degeneration of Wharton's jelly or pseudocysts.[56-63] A true incidence of these lesions is unknown, though a prevalence of 3.4% was noted in study that sonographically assessed first trimester pregnancies.[62] The umbilical cord cysts were observed in 29 of the 859 pregnancies at 7-13 weeks of gestation, and in more than 20% of cases fetal chromosomal or structural defects were noted. During the antenatal fetal assessment, majority of these lesions are observed near the fetal abdominal cord insertion, or at the insertion of the cord into the placenta. If the hyperechogenic mass of the cord is noted, it is likely hemangioma or angiomyxomas that usually arise from proliferation of the primitive angiogenic mesenchyme. It is important to note that these lesions are frequently associated with elevated maternal serum alpha-protein levels.[63] The prognosis and management of these pregnancies largely vary on associated fetal anomalies and chromosomal aberration if present. If the mass is large, there is always possibility of compression of the umbilical cord vessels that could modify fetal growth pattern and even cause fetal nonimmune hydrops. Therefore, serial ultrasound for reassessment of the umbilical cord lesions and interval fetal growth appears to be reasonable modification of the obstetrical management.

SUMMARY

In conclusion, we believe that at present time an evaluation of the umbilical cord, not only for number of vessels, but rather for cord thickness or cross-sectional area, an amount of Wharton's jelly, abnormal umbilical cord coiling, together with the assessment of the umbilical cord arteries' blood flow characteristics, may provide valuable information about the pregnancy and fetal well-being. The assessment of the blood flow patterns remain as an important diagnostic tool in analyzing of the fetus with growth restriction.

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