

ULTRASOUND DIAGNOSIS AND MANAGEMENT OF UMBILICAL CORD ABNORMALITIES

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SUMMARY

The incidence of placenta and umbilical cord abnormalities is high in abnormal course of the delivery. Although the detection rate of umbilical cord abnormalities is steadily increasing with the improvement of ultrasound technology, and the fact that ultrasound scanning can distinguish umbilical cord conditions, this information has not exerted much impact on the management of labor to date. We believe prenatal detection of umbilical cord abnormalities must reduce the number of emergent cesarean sections and intrauterine fetal death. In this review, we describe the ultrasound diagnosis and management of major umbilical cord abnormalities, including abnormalities of cord insertion site (velamentous and marginal cord insertion), hypercoiled cord and nuchal cord, considering the current knowledge on physiologic and pathologic aspects of each umbilical cord abnormality. [*Taiwan J Obstet Gynecol* 2009;48(1):23-27]

Key Words: fetal heart rate, hypercoiled cord, nuchal cord, ultrasound, umbilical cord, velamentous cord insertion

Abnormal Cord Insertion of the Placenta

The umbilical cord normally inserts into the central portion of the placenta, well away from the placental edge. Velamentous cord insertion (VCI) is an abnormal cord insertion (CI) in which the umbilical vessels diverge as they traverse between the amnion and chorion before reaching the placenta. Marginal CI is an abnormal CI in which the umbilical cord inserts into the placental edge. The rate of VCI ranges from 0.5% to 1.69% in singleton pregnancies, and the prevalence of VCI is 10-fold higher in multiple pregnancies than in singleton pregnancies [1,2]. It has been reported that abnormal CI is associated with fetal growth restriction, preterm labor, abnormal intrapartum fetal heart rate pattern, low Apgar scores at 1 and 5 minutes, neonatal death [1,2], and abruption of the placenta [3-6]. Especially, in cases which has the CI site on the lower uterine segment, VCI is strongly associated with variable decelerations

(VDs), non-reassuring fetal status, emergent cesarean sections, and other perinatal complications [6]. Some vascular and placental structural abnormalities complicated with lower VCI may be associated with extension of the lower uterine segment and atrophy of the chorion villosum that covers the lower segment of the uterus [6].

Abnormal fetal heart rate patterns and some perinatal complications are caused by lack of Wharton's jelly, which results in compression of vessels of VCI during uterine contraction or fetal movement. Aberrant vessels in lower VCI tended to be longer than those in middle and upper VCI [6,7]. It seems that the longer aberrant vessels are readily compressed by the fetal head in the lower uterine segment, which results in an abnormal fetal heart rate pattern and intrapartum complications during labor.

Vasa previa is a form of VCI in which the velamentous vessels traverse the fetal membranes on or near the internal os. The incidence of vasa previa is estimated to be about 1 in 1,200-5,000 pregnancies [8-11]. Vasa previa has unsupported fetal vessels below the fetal presenting part, so these fetal vessels are easily compressed or ruptured when uterine contractions or membrane rupture occurs, resulting in fetal exsanguination. Intrapartum clinical diagnosis is rarely made and the



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diagnosis is very difficult [12]. Oyelese et al [13] performed a multicenter study of 155 pregnancies complicated by vasa previa. Between 1991 and 2003, 61 of 155 cases were prenatally diagnosed by ultrasonography/color Doppler, and the infant survival rate was 97% (59/61). In the cases not prenatally diagnosed, the survival rate was 44% (41/94). Furthermore, multivariable logistic regression analysis revealed that the significant predictors of neonatal survival were prenatal diagnosis and gestational age at delivery. The high fetal mortality due to vasa previa can be reduced by antenatal diagnosis and elective cesarean section [10,12–14].

Abnormal placentation and situations sometimes lead to be risk factor of vasa previa and VCI [15]. Previous studies have reported that the risk factors for vasa previa include the ultrasound diagnosis of low-lying placenta or placenta previa at earlier gestation [9,11,16,17], a bilobed or succenturiate placenta [9,11,16,17], multiple gestation [17], suspicion of aberrant vessels [17], VCI [17,18], CI into lower uterine segment [19], and an *in vitro* fertilization pregnancy [13,15].

Ultrasound Diagnosis of the Abnormal CI

As visualization of the placental CI site becomes more difficult with advancing gestation, the placental CI site should be evaluated in the mid-trimester [10,14,20]. Our criteria for ultrasound diagnosis of VCI are: (1) umbilical vessels enter the placenta margin parallel to the uterine wall and connect to superficial placental vessels; (2) the CI is immobile, even when the uterus is shaken; and (3) the umbilical vessels diverge as they traverse the membrane. In fact, the CI site was determined less frequently in the cases of marginal CI and VCI than in the normal CI in our previous study [6]. Existence of an abnormal CI should be strongly suspected when it is difficult to image the CI site (decreased sensitivity); thus, a more precise scan (scanning in different body positions and using color Doppler) is indicated in such cases.

Additional transvaginal color Doppler sonography of the cervical region has been recommended to detect vasa previa and lower VCI during the third trimester for women with the aforementioned increased risk [13,16].

In cases in which CI was noted to be in the lower uterine segment during the first trimester, such developmental abnormalities of the placenta and the umbilical cord occurred frequently as low-lying placentas, infarction of placentas, abruption of placentas, VCI, and marginal CI [21]. Thus, it may be useful to perform the systematic identification of CI on the lower uterine segment during the first trimester for identification of high-risk pregnancies. In low CI cases, reevaluation

during the second and third trimester by using color Doppler ultrasonography is recommended.

Hypercoiled Cord

Although the umbilical cords and their blood vessels are necessary for the survival of the fetus, umbilical blood vessels are so vulnerable to kinking, compression, traction and torsion. A coiled umbilical cord with the support of Wharton's jelly is thought to be more resistant to torsion, stretch, and compression [22]. However, several studies have addressed that hypercoiled cords (HCCs) are correlated with poor perinatal outcome such as low birth weight and meconium staining of amniotic fluid at birth, and fetal growth restriction [23–28]. Previous studies suggest that extremely coiled umbilical vessels are less flexible or more prone to kinking and torsion during labor, leading to fetal hypoxia [26,27]. Unfortunately, the cause of umbilical vascular coiling is unknown. Hypotheses include fetal movement, active or passive torsion of the embryo, differential umbilical vascular growth rates, fetal hemodynamic forces, and the arrangement of muscular fibers in the umbilical wall [23,29]. The umbilical coiling index (UCI) is calculated by dividing the total number of coils by the length of the cord in centimeters after delivery. HCC is defined in cases of umbilical coiling index ≥ 0.3 coils/cm [27].

Ultrasound Diagnosis of the HCC

The UCI was antenatally calculated by measuring the distance between two adjacent coils of umbilical artery from the right outer surface of the vascular wall to its next twist (antenatal UCI = 1/distance in centimeters) as proposed by Degani et al [22]. It is known that antenatal UCI is higher than the postnatal UCI (0.44 ± 0.11 vs. 0.28 ± 0.08 ; $p < 0.001$) [22]. It has also been reported that values of antenatal UCI in the second trimester were 0.602, 0.403 and 0.204 in 90th percentile, mean and 10th percentile, respectively [24]. We usually diagnose a case as HCC when antenatal UCI is above 0.6. However, only a few fetuses whose umbilical cord was antenatally diagnosed as HCC would be compromised during pregnancy and even delivery. It is necessary to actually pick up high-risk cases of HCC among them.

There have been some reports of HCC associated with umbilical blood flow. Predanic et al [30] noted that increased umbilical coiling was associated strongly with increased umbilical vein blood flow (lower resistance

index value). On the other hand, HCC is associated with a pulsate pattern of the umbilical venous flow velocity waveforms similar to those seen in abnormalities of the fetal central venous flow, secondary to severe circulatory compromise [31,32]. However, there are no predictable methods of non-reassuring fetal status or intrauterine fetal death.

From clinical data of our department, umbilical cord abnormalities accounted for 45% of causes of intrauterine fetal death [33]. Among these cases with cord abnormalities, HCCs were observed in more than 50%. We frequently observe narrow and weak cords near the fetal side in intrauterine fetal death cases with HCC. It is supposed that the cord of fetal side is weakest in the whole umbilical cord, and that this state of severe HCC is associated with sudden fetal death. Skulstad et al [34–36] evaluated prenatally umbilical venous velocities and umbilical ring constrictions. They demonstrated that blood velocity was higher in the umbilical vein at abdominal wall than the cord and concluded that measurement of vessel diameters at the umbilical ring was too small to be valid, but the measurement of the high blood velocity or the increase in velocity was a better marker for vascular constriction at the umbilical ring than the direct diameter measurement.

For better management of HCC, it is recommended to diagnose coiling index in mid-gestation and to follow up the case using various Doppler measurements of fetal and umbilical blood flow.

Nuchal Cord

Nuchal cord (NC) is most frequently seen among umbilical cord abnormalities. The prevalence has been reported to be 15–24% at delivery [37,38]. In a large population study (166,318 deliveries) by Sheiner et al [37], NC was not associated with adverse perinatal outcome. Ogueh et al [38] investigated 57,853 deliveries and reported that NC was associated with induction of labor, slow progress of labor, and shoulder dystocia. Several studies were conflicting in the incidence of cesarean section due to presence of NC [39–42]. On the other hand, Larson et al [39] observed that the pregnancy outcomes in cases of multiple NC entanglement, compared with a single or absent cord entanglement, were more likely to exhibit an abnormal fetal heart rate pattern during advanced labor, to require low- or mid-forceps application, and to have meconium, a low 1-minute Apgar score and an umbilical artery pH ≤ 7.10 . Although the management of multiple NC is arguable, single NC does not seem to influence the clinical management.

Ultrasound Diagnosis of NC

NCs are usually visualized ultrasonically as dimples with umbilical cords at the neck of the fetus on sagittal view. They should be identified by presence of the cord in the transverse and sagittal planes of the neck and lying around at least three of the four sides of the neck. Although there appears to be a linear increase over gestation in the presence of both single and multiple loops [43–45], NC keeps appearing and disappearing over time [45]. The difficulty encountered in visualizing the NC at term and prior to induction of labor may be due to fetal crowding, low position of the fetal head or reduced amniotic fluid volume [42]. Generally, the sensitivity of diagnosis is higher with color Doppler imaging, and it may have a particular advantage in the presence of ruptured membranes [42].

Fetal Heart Rate Patterns in Cases with Umbilical Cord Abnormalities

VDs are most frequently observed as a type of periodic change in fetal heart rate monitoring and are likely caused by cord occlusion. Cord occlusion, either partial or complete, can cause both increases in afterload and decreases in fetal arterial oxygen content, both of which will result in an activated vagal reflex causing bradycardia [46]. We reported that in the first stage of labor, frequencies of VDs were $34.5\% \pm 23.8\%$, $27.3\% \pm 25.5\%$ and $20.2\% \pm 22.8\%$ in cases with VCIs, HCCs and NCs, respectively. These were significantly higher than in controls ($11.7\% \pm 17.3\%$; $p < 0.0001$) [47]. We also reported that the frequencies of VDs were high in the first stage of labor in the presence of cord abnormalities, although frequencies of VDs were not different from controls in the second stage of labor. This finding suggests that even in cases without cord abnormality, VDs occur frequently in the second stage, which is because of not only cord compression but also significant head compression. Head compression causes a vagal discharge due to dural stimulation resulting in bradycardia which can be diagnosed as VD [46].

Atypical VD was reported by Krebs et al [48] in 1983 as prognostically unfavorable with features indicative of fetal hypoxia, including slow return of the fetal heart rate to the baseline, loss of variability during the deceleration, loss of initial and/or secondary accelerations, persistence of secondary acceleration (overshoot), continuation of the fetal heart rate at a lower level, and biphasic deceleration. In our study, atypical VDs frequently occurred in cases with VCI, HCC and NC in the first stage of labor, although frequencies of both atypical

and typical VDs were not different compared with controls in the second stage of labor [49]. Moreover, in NC cases, a mixed VD pattern (typical and atypical) was observed from the first stage of labor onward [49]. Atypical VD in the first stage of labor may be characteristic fetal heart rate pattern for VCI and HCC. Appearance of recurrent atypical VDs during labor at the first stage in cases with antepartum information of these umbilical cord abnormalities could be a foresight of unfavorable fetal status. Among atypical VDs, frequency of loss of initial and/or secondary accelerations (VD with no acceleration [VDna]) was significantly higher than the control in our same study [49]. Lee et al [50] suggested that variation in typical VD is caused by different degrees of partial cord compression. As a specific state, the cause of frequent VDs in VCI cases is the compression of aberrant vessels and that the blood flows of both arteries and veins would be obstructed at the same time by the uterine contraction. In cases of HCC, umbilical cords are less flexible or more prone to kinking and torsion [26,27], and narrow and weak cords near the fetal side can tend to obstruct the flows of umbilical vessels at the same time. Thus, VD without baroreceptor-mediated acceleration (VDna) might to be a characteristic fetal heart rate pattern in VCI and HCC cases [7,49].

In cases of VCI and vasa previa, frequent fetal heart rate monitoring in the late third trimester is required. Patients should be educated regarding signs and symptoms of preterm labor. Oyelese et al [13] recommended that women with prenatally diagnosed vasa previa be offered elective delivery by cesarean section at about 35 weeks of gestation or earlier if fetal lung maturity is documented. As for VCI, we recommend elective cesarean section in cases with lower VCI, because we consider that lower VCI is analogous to vasa previa and has very high rate of non-reassuring fetal status. Alternatively, other VCI cases can be offered attempted vaginal delivery with the backup of emergent cesarean delivery. During labor, VD, especially VDna, should be considered a warning sign of abrupt fetal compromise.

Conclusion

Antenatal ultrasound diagnosis of umbilical cord abnormalities may be helpful in picking up cases in which strict fetal monitoring is warranted antenatally as well as during labor. This information could then be used to triage patients to either high-risk or low-risk cases and could alert the physician to the increased risk of non-reassuring fetal status. In cases with cord abnormalities, especially VCIs and HCCs, intensive monitoring

and preparation for cesarean delivery are indicated. We do believe that the neonatal outcome must be improved by these precise managements.

References

1. Eddleman KA, Lockwood CJ, Berkowitz GS, Lapinski RH, Berkowitz RL. Clinical significance and sonographic diagnosis of velamentous umbilical cord insertion. *Am J Perinatol* 1992; 9:123-6.
2. Heinonen S, Ryyanen M, Kirkinen P, Saarikoski S. Perinatal diagnostic evaluation of velamentous umbilical cord insertion: clinical, Doppler, and ultrasonic findings. *Obstet Gynecol* 1996;87:112-7.
3. Ananth CV, Savitz DA, Williams MA. Placental abruption and its association with hypertension and prolonged rupture of membranes: a methodologic review and meta-analysis. *Obstet Gynecol* 1996;88:309-18.
4. Ananth CV, Smulian JC, Vintzileos AM. Incidence of placental abruption in relation to cigarette smoking and hypertensive disorders during pregnancy: a meta-analysis of observational studies. *Obstet Gynecol* 1999;93:622-8.
5. Toivonen S, Heinonen S, Anttila M, Kosma VM, Saarikoski S. Reproductive risk factors, Doppler findings, and outcome of affected births in placental abruption: a population-based analysis. *Am J Perinatol* 2002;19:451-60.
6. Hasegawa J, Matsuoka R, Ichizuka K, Sekizawa A, Farina A, Okai T. Velamentous cord insertion into the lower third of the uterus is associated with intrapartum fetal heart rate abnormalities. *Ultrasound Obstet Gynecol* 2006;27:425-9.
7. Hasegawa J, Matsuoka R, Ichizuka K, Sekizawa A, Farina A, Okai T. Velamentous cord insertion and atypical variable decelerations with no accelerations. *Int J Gynaecol Obstet* 2005;90:26-30.
8. Pent D. Vasa previa. *Trans Pac Coast Obstet Gynecol Soc* 1979; 46:50-4.
9. 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.
10. Nomiyama M, Toyota Y, Kawano H. Antenatal diagnosis of velamentous umbilical cord insertion and vasa previa with color Doppler imaging. *Ultrasound Obstet Gynecol* 1998;12: 426-9.
11. Oyelese KO, Turner M, Lees C, Campbell S. Vasa previa: an avoidable obstetric tragedy. *Obstet Gynecol Surv* 1999;54: 138-45.
12. Catanzarite V, Maida C, Thomas W, Mendoza A, Stanco L, Piacquadio KM. Prenatal sonographic diagnosis of vasa previa: ultrasound findings and obstetric outcome in ten cases. *Ultrasound Obstet Gynecol* 2001;18:109-15.
13. Oyelese Y, Catanzarite V, Prefumo F, et al. Vasa previa: the impact of prenatal diagnosis on outcomes. *Obstet Gynecol* 2004;103:937-42.
14. Sepulveda W, Rojas I, Robert JA, Schnapp C, Alcalde JL. Prenatal detection of velamentous insertion of the umbilical cord: a prospective color Doppler ultrasound study. *Ultrasound Obstet Gynecol* 2003;21:564-9.

15. Schachter M, Tovbin Y, Arieli S, Friedler S, Ron-El R, Sherman D. In vitro fertilization is a risk factor for vasa previa. *Fertil Steril* 2002;78:642-3.
16. Francois K, Mayer S, Harris C, Perlow JH. Association of vasa previa at delivery with a history of second-trimester placenta previa. *J Reprod Med* 2003;48:771-4.
17. Lee W, Lee VL, Kirk JS, Sloan CT, Smith RS, Comstock CH. Vasa previa: prenatal diagnosis, natural evolution, and clinical outcome. *Obstet Gynecol* 2000;95:572-6.
18. Pretorius DH, Chau C, Poeltler DM, Mendoza A, Catanzarite VA, Hollenbach KA. Placental cord insertion visualization with prenatal ultrasonography. *J Ultrasound Med* 1996;15:585-93.
19. Hasegawa J, Matsuoka R, Ichizuka K, Fujikawa H, Sekizawa A, Okai T. Umbilical cord insertion to the lower uterine segment is risk factor for vasa previa. *Fetal Diagn Ther* 2007;22:358-60.
20. Oyelese Y. Placenta previa and vasa previa: time to leave the Dark Ages. *Ultrasound Obstet Gynecol* 2001;18:96-9.
21. Hasegawa J, Matsuoka R, Ichizuka K, Otsuki K, Sekizawa A, Farina A, Okai T. Cord insertion into the lower third of the uterus in the first trimester is associated with placental and umbilical cord abnormalities. *Ultrasound Obstet Gynecol* 2006;28:183-6.
22. Degani S, Lewinsky RM, Berger H, Spiegel D. Sonographic estimation of umbilical coiling index and correlation with Doppler flow characteristics. *Obstet Gynecol* 1995;86:990-3.
23. de Laat MW, Franx A, Bots ML, Visser GH, Nikkels PG. Umbilical coiling index in normal and complicated pregnancies. *Obstet Gynecol* 2006;107:1049-55.
24. Predanic M, Perni SC, Chasen ST, Baergen RN, Chervenak FA. Ultrasound evaluation of abnormal umbilical cord coiling in second trimester of gestation in association with adverse pregnancy outcome. *Am J Obstet Gynecol* 2005;193:387-94.
25. Ezimokhai M, Rizk DE, Thomas L. Maternal risk factors for abnormal vascular coiling of the umbilical cord. *Am J Perinatol* 2000;17:441-5.
26. Rana J, Ebert GA, Kappy KA. Adverse perinatal outcome in patients with an abnormal umbilical coiling index. *Obstet Gynecol* 1995;85:573-7.
27. Strong TH Jr, Jarles DL, Vega JS, Feldman DB. The umbilical coiling index. *Am J Obstet Gynecol* 1994;170:29-32.
28. Ercal T, Lacin S, Altunyurt S, Saygili U, Cinar O, Mumcu A. Umbilical coiling index: is it a marker for the foetus at risk? *Br J Clin Pract* 1996;50:254-6.
29. Strong TH Jr, Elliott JP, Radin TG. Non-coiled umbilical blood vessels: a new marker for the fetus at risk. *Obstet Gynecol* 1993;81:409-11.
30. Predanic M, Perni SC, Chervenak FA. Antenatal umbilical coiling index and Doppler flow characteristics. *Ultrasound Obstet Gynecol* 2006;28:699-703.
31. Nakai Y, Imanaka M, Nishio J, Ogita S. Umbilical venous pulsation associated with hypercoiled cord in growth-retarded fetuses. *Gynecol Obstet Invest* 1997;43:64-7.
32. Nakai Y, Imanaka M, Nishio J, Ogita S. Umbilical venous pulsation and regional circulatory disturbance. *Ultrasound Med Biol* 1997;23:1165-9.
33. Hasegawa J. Studies for early identification of umbilical cord abnormalities and for managements of high risk pregnancies with them. *Acta Obstet Gynaecol Jpn* 2008;60:1723-34.
34. Skulstad SM, Rasmussen S, Iversen OE, Kiserud T. The development of high venous velocity at the fetal umbilical ring during gestational weeks 11-19. *BJOG* 2001;108:248-53.
35. Skulstad SM, Rasmussen S, Seglem S, Svanaes RH, Aareskjold HM, Kiserud T. The effect of umbilical venous constriction on placental development, cord length and perinatal outcome. *Early Hum Dev* 2005;81:325-31.
36. Skulstad SM, Kiserud T, Rasmussen S. Degree of fetal umbilical venous constriction at the abdominal wall in a low-risk population at 20-40 weeks of gestation. *Prenat Diagn* 2002;22:1022-7.
37. Sheiner E, Abramowicz JS, Levy A, Silberstein T, Mazor M, Hershkovitz R. Nuchal cord is not associated with adverse perinatal outcome. *Arch Gynecol Obstet* 2006;274:81-3.
38. Ogueh O, Al-Tarkait A, Vallerand D, Rouah F, Morin L, Benjamin A, Usher RH. Obstetrical factors related to nuchal cord. *Acta Obstet Gynecol Scand* 2006;85:810-4.
39. Larson JD, Rayburn WF, Crosby S, Thurnau GR. Multiple nuchal cord entanglements and intrapartum complications. *Am J Obstet Gynecol* 1995;173:1228-31.
40. Jauniaux E, Ramsay B, Peellaerts C, Scholler Y. Perinatal features of pregnancies complicated by nuchal cord. *Am J Perinatol* 1995;12:255-8.
41. Rhoades DA, Latza U, Mueller BA. Risk factors and outcomes associated with nuchal cord: a population-based study. *J Reprod Med* 1999;44:39-45.
42. Peregrine E, O'Brien P, Jauniaux E. Ultrasound detection of nuchal cord prior to labor induction and the risk of Cesarean section. *Ultrasound Obstet Gynecol* 2005;25:160-4.
43. Larson JD, Rayburn WF, Harlan VL. Nuchal cord entanglements and gestational age. *Am J Perinatol* 1997;14:555-7.
44. Clapp JF 3rd, Stepanchak W, Hashimoto K, Ehrenberg H, Lopez B. The natural history of antenatal nuchal cords. *Am J Obstet Gynecol* 2003;189:488-93.
45. Lal N, Deka D, Mittal S. Does the nuchal cord persist? An ultrasound and color-Doppler-based prospective study. *J Obstet Gynaecol Res* 2008;34:314-7.
46. Ball RH, Parer JT. The physiologic mechanisms of variable decelerations. *Am J Obstet Gynecol* 1992;166:1683-8; discussion 8-9.
47. Hasegawa J, Ichizuka K, Matsuoka R, Masamitsu N, Sekizawa A, Okai T. Do fetal heart rate deceleration patterns during labor differ between various umbilical cord abnormalities? *J Perinat Med* (In press).
48. Krebs HB, Petres RE, Dunn LJ. Intrapartum fetal heart rate monitoring, VIII: atypical variable decelerations. *Am J Obstet Gynecol* 1983;145:297-305.
49. Hasegawa J, Matsuoka R, Ichizuka K, et al. Atypical variable deceleration in the first stage of labor is a characteristic fetal heart-rate pattern for velamentous cord insertion and hypercoiled cord. *J Obstet Gynaecol Res* 2009;35:35-9.
50. Lee CY, Di Loreto PC, O' Lane JM. A study of fetal heart rate acceleration patterns. *Obstet Gynecol* 1975;45:142-6.