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Review

Umbilical cord accidents and legal implications

Jason H. Collins*

Pregnancy Institute, P.O. Box 806, New Roads, LA 70760, USA

S U M M A R Y

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Umbilical cord accidents (UCA) are a significant cause of stillbirth. Although infrequent, litigation may occur when there is a poor outcome associated with UCA. With advances in imaging, the ability to identify UCA by ultrasound and magnetic resonance imaging raises awareness of the risk of a poor outcome. Management of a pregnancy with an identified UCA may require more fetal surveillance by both the mother and caregiver. This is especially important if there is a previous history of UCA with or without stillbirth. UCA should be an acknowledged risk which is part of prenatal screening. In the event of a poor outcome associated with UCA, it is recommended that the patient be fully informed of all prenatal information including images. Excellent communication with parents who are looking for answers after a tragic outcome may help to decrease litigation risk.

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1. Introduction

In evolutionary terms, the umbilical cord is an ancient structure with many functions. Melbourne Museum has a 375-million-year-old placoderm fish fossil showing an embryo attached to its mother by an umbilical cord [1]. The umbilical cord enables the fetus to develop, grow, adapt, survive, and be born. It is then discarded. Umbilical cord tissues are differentiated by genetics, biochemistry, function, and cell type. This makes the human umbilical cord not only an organ but a disposable one. It is of fetal origin and may be paternally influenced [2]. It can contain mosaic genetics different from the placenta. Umbilical cord tissue with mosaic trisomy 2, 6, 14, and 15 have all been described [3].

In a review of 16 body-stalk anomalies in which the umbilical cord was very short or absent, normal karyotypes were found. Most infants were stillborn. To date there are no specific studies pointing to the genetic origin of the human umbilical cord. There is a question of the role of viral infection and the environment in disturbing the embryonic origin and development of the umbilical cord [4–6].

Embryonic studies point to the origin of the umbilical cord in an area located at the lower third of the conceptus known as the primitive ridge. The umbilical cord begins at the proximal portion at the 'Allantoic Core Domain'. Between four and six weeks, as the embryonic disk takes a cylindrical shape, the umbilical cord 'grows'

while fusing with the placenta and its vessels. The umbilical cord elongates away from the placenta, increasing in diameter and forming umbilical cord vessels [7]. The average umbilical cord grows, elongates in the middle, and has three or four helices at the fetal end with a central straighter section and three or four helices toward the placental end. There are eight different umbilical cord forms, but the genetics have yet to be studied. The umbilical cord is not placental in origin but fetal. Scientifically it should be treated as a separate organ of reproduction. The proximal fetal attachment is unique and develops a sac (herniation) by 10 weeks. This area houses the intestines until the 12th week of gestation. At this time, the umbilical cord is short, usually shorter than the head-to-tail (crown–rump) length of the embryo and of proportionately large diameter which does not tolerate rotation about itself. As the umbilical cord elongates, the proximal cord encompassing the intestinal pouch cannot be disturbed. The distal initial stalk develops in the center of the placental implantation site. The allantois begins at the pole (end) of the embryo and eventually centers itself in the fetus. By 10–12 weeks, the intestines leave the proximal cord and return to the abdominal cavity. The elongation of the cord begins with umbilical vein and artery growth and the development of Wharton's jelly.

Umbilical cord properties such as tensile strength, diameter, circumference, weight, and length are determined genetically. The anatomical connection of the umbilical cord to the placenta and fetus is specialized. If defective, fetal loss results. The umbilical cord varies in its microstructure, nerve content, and arterial and venous relationship. It is important to be aware of these elements when providing prenatal care, as well as to record observations of the

* Tel.: +1 985 710 0672.

E-mail address: jcollinsmd77@gmail.com.

umbilical cord structure after the delivery [8]. Documenting a thin cord, long cord (>80 cm), short cord (<35 cm), overly twisted cord (torsion), straight cord, discoloration with meconium, funisitis, growths, hemorrhages, etc. may help determine the cause of stillbirth in a medico-legal discussion.

The Stillbirth Collaborative Research Network recently reported on the probable or possible cause of death of 512 stillbirths whose mothers consented to complete postmortem examination [9]. Umbilical cord accidents were found in 10% of stillbirths. In Caucasians, the UCA-associated stillbirth rate was 13% and in non-Hispanic black patients it was 4%. A literature review places the UCA-associated stillbirth rate at 15%. These databases do not include stillbirth secondary to several UCA pathologic conditions such as torsion, multiple cord entanglement, and abnormal placental cord insertion. The main reason for these exclusions is the belief that these abnormalities do not cause morbidity, actual death, or recurrent stillbirth. There is ample peer-reviewed literature to refute this [10–16], a finding of considerable medico-legal significance. Laymen, who make up a jury, generally agree that umbilical cord compression may cause death.

There are many stillbirth studies which have accumulated since the 1950s. One of the largest, The National Perinatal Collaborative Project, reviewed more than 50,000 pregnancies [17]. One pathologic finding was that a 'long umbilical cord' is a risk factor for stillbirth. Since this landmark study, other umbilical cord pathologies have been documented as risk factors for stillbirth [18,19]. In a recent report reviewing 121 stillbirths, umbilical cord pathology was associated with third-trimester stillbirth in 33.3% of cases [20]. Placental pathology association with stillbirth has also been well described [21–26]. In a review of 520 consecutive stillbirths, placental vasculopathies were felt to support occult umbilical cord occlusion as the cause of death [27]. For medico-legal purposes it is highly recommended that any stillbirth or finding of an umbilical cord-related event at delivery prompt a placental pathology review. As Horn et al. note, 'especially in the field of litigation processes, placental morphology may be very important for the demonstration that, for example, brain damage or intrauterine death resulted from placental or umbilical cord pathology and not from physician failure' [28].

One of the first published accounts of a UCA in western medical writings was by William Smellie in his 'Treatise on midwifery' in 1750. One of the earliest published drawings of a UCA was by Andrew Bell in the Encyclopedia Britannica in 1769, depicting a fetal death with a combination of one nuchal cord, a body loop, and a true knot. There are more than 100 published animal model studies showing various sequences of umbilical cord compression leading to fetal morbidity and mortality.

An increasing focus is being placed on screening for and monitoring conditions that may place a fetus at increased risk of UCA. The American College of Obstetricians and Gynecologists recommends a fetal review for a heart rate of 90 beats/min for 1 min on a recorded non-stress test. The 18–20-week ultrasound review should include the umbilical cord, its characteristics, and a description of its placental and fetal attachments. The American Association of Ultrasound Technologists has defined these parameters for umbilical cord abnormalities:

- abnormal insertion
- vasa previa
- abnormal composition
- cysts, hematomas, and masses
- umbilical cord thrombosis
- coiling, collapse, knotting and prolapse.

The umbilical cord may be sonographically evaluated. This includes the appearance, composition, location, and size of the cord.

These findings should be documented [29,30]. A normal cord has a single vein and two arteries that have a helical, rope-like appearance with an average length of 55 cm. The human umbilical cord has eight forms. The common form of two helical arteries around a central vein has an average of three twists and eight helices. Twists and helices may be differentiated. Twists can be untwisted. Helices are permanent and cannot be untwisted (Fig. 1).

This is helpful to note at delivery of a stillbirth. To date, this detail is not included in most studies on stillbirth, yet could be invaluable in a medico-legal review. Over-twisting is called torsion. The pattern implies above-average repositioning of a fetus and may be a possible cause of death. In the animal world, torsion is a common cause of foal deaths. Absence of twisting (straight cord) often is associated with a decrease in fetal movement and a poor pregnancy prognosis. The 'normal umbilical cord' was first described by William Hunter in the 1700s and it was noted that the average cord had 11 turns (eight helices and three twists).

Umbilical cord pathology is separate from placental pathology. It is becoming possible to distinguish between umbilical cord compression causing placental change leading to umbilical cord changes. The fetus may exhibit behavioral change from placental compromise, sometimes in the form of fetal repositioning, which may lead to umbilical cord entanglement, torsion, or compression. Small-for-gestational age (SGA) fetuses may lead to UCA, which may lead to SGA changes (umbilical cord constriction). Look for UCA with ultrasound once it is noted that a fetus has changed from a normal weight or behavior. Current knowledge of the human umbilical cord and its influence on the fetus is limited. For this reason, it may be prudent to evaluate the umbilical cord following even minor maternal complaints. Interactions between the fetus and umbilical cord are becoming apparent on studies of fetal behavior. Fetal body movements have been studied with 24 h ultrasound. These movements are unique between midnight and 06:00. Time of fetal behavioral observation may need to be included in any future stillbirth study [31–35]. Fetal behavioral issues have medico-legal significance. A recent case regarding decreased fetal movements has been described [36]. The case reported changes in the fetal heart rate after decreased fetal movement. Patients who report change in fetal behavior should be evaluated. Fifteen percent of patients will report decreased fetal movement. These patients are often dismissed or told to consume a sweet drink, despite there being no published information that supports this management [37].

Hyperactivity is a fetal response associated with risk factors for umbilical cord compression. It may be related to intrauterine umbilical blood flow disturbances, which stimulates the fetus to react reflexively and excessively. Animal studies have reproduced forms of hyperactivity with cord compression. Hyperactivity may be a prenatal behavior capable of repositioning the fetus and relieving umbilical cord compression. In the rat model, umbilical cord compression triggered lateral trunk curls, head tosses, and foreleg extensions. In the sheep model, intermittent umbilical cord compression triggered fetal hiccups. Hiccups occurring daily after 28 weeks, and more than four times per day, may benefit from fetal evaluation.

Hypoactivity may indicate fetal compromise, and complaints should be investigated promptly, with consideration of fetal heart rate testing and possibly ultrasound [38–40].

Recent research into circadian rhythms may help explain why UCA stillbirth often occurs between 02:00 and 04:00. Melatonin stimulates uterine contractions through the **MT2** receptor. Melatonin secretion from the pineal gland begins around 22:00 and peaks to 60 pg at 03:00. Serum levels decline to <10 pg by 06:00. Uterine stimulation intensifies as a result and may be overwhelming to a compromised fetus, especially one experiencing

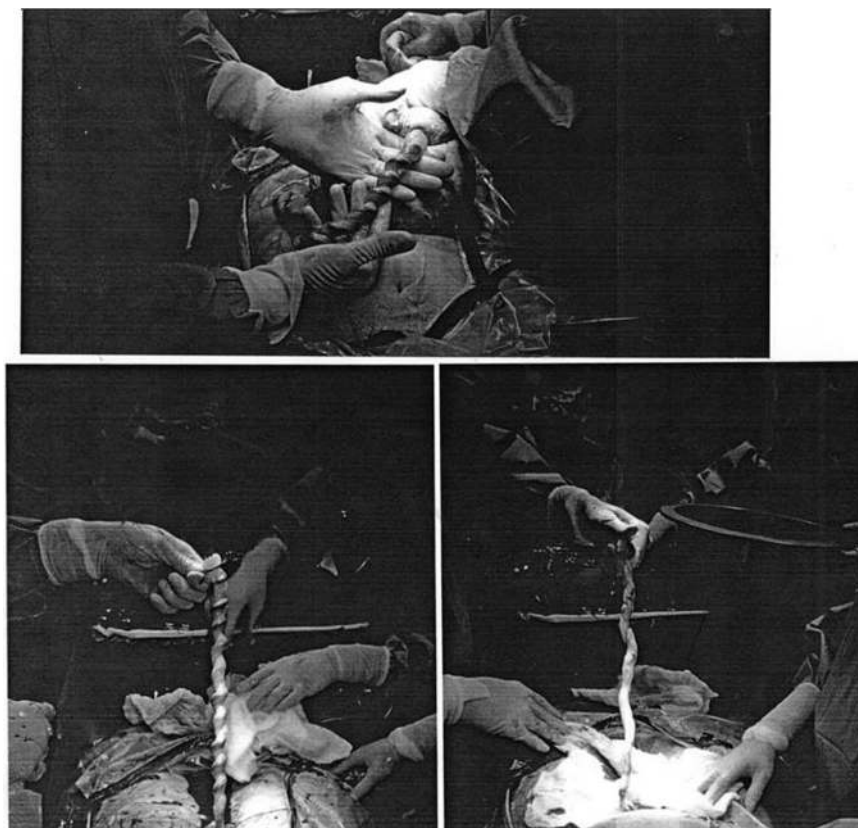


Fig. 1. Umbilical cord torsion in a liveborn at cesarean section. Upper: torsion umbilical cord accident. Lower left: multiple twists. Lower right: untwisted eight times [63].

intermittent umbilical cord compression. Pregnancy Institute has documented more than 1000 UCA stillbirths through patient interview that occurred during maternal sleep. These interviews continue on the average of one per week by e-mail, phone, or direct conversation. As of this writing, a neonatologist lost her second baby to a UCA at 32 weeks. She was told by her obstetrician at the beginning of her second pregnancy that a second loss could never happen. Her baby died of a UCA during maternal sleep (personal communication).

Circadian-associated stillbirth, especially when secondary to UCA, is an important part of explaining a sequence of events leading to fetal death. Patients reported a normal biophysical profile at 18:00 and/or an NST the evening before demise. They reportedly understood that the chance of detecting a compromised baby between 00:00 to 06:00 was not from caregiver error. This explanation was used in two cases where the author was a defense expert witness.

Several e-mails describing cases of loss can be found online at www.preginst.com. It is particularly disturbing when these patients are medical professionals. Like all mothers, they want to know what caused the stillbirth. The following suggestions are examples of what to say and what not to say, in part for medico-legal reasons, when patients inquire about a UCA stillbirth:

I. What was the cause of death? Not known but probably not umbilical-cord-related [41].

Instead: An umbilical cord issue was observed and a placental pathology report is needed to be certain of an association. An autopsy would be helpful.

II. Can ultrasound detect UCA? No, ultrasound cannot see the umbilical cord (a frequent response).

Instead: Yes, ultrasound can visualize the umbilical cord and we will review the images on record (many of these patients have gone to 'boutique' ultrasound facilities and usually send copies with their e-mail).

III. Can UCA happen again? No, there is a one in a million chance it will happen again (also a frequent comment).

Instead: Stillbirth has a five-fold risk of recurrence and UCA can unfortunately recur in the same mother in a subsequent pregnancy, as illustrated in the following case report:

'During a span of 3.5 years, a 30-year-old, gravida 9, para 3 woman experienced three pregnancies complicated by umbilical cord torsion and constriction. In each case, the complication resulted in acute vascular compromise and intrauterine fetal demise. Gross examination disclosed cord constriction and torsion at the fetal end of the cord in each instance. Histologic sections from the cord torsion sites demonstrated fibrosis and deficiencies in Wharton's jelly in each case. Cytogenetic studies prepared using fetal villous tissue demonstrated normal karyotypes in fetal cells from the first two pregnancies (46 XX and 46 XY, respectively). The karyotype from the third pregnancy showed a 46 XX,del (X)(q24) mutation in three of 15 cultured cells, while 12 of 15 cells possessed a normal 46 XX karyotype. This cytogenetic abnormality was not believed to represent the cause of fetal demise in this case. This is probably the first report of umbilical cord torsion in three pregnancies within one family. The familial clustering observed in this report suggests that a genetic predisposition for umbilical cord torsion may exist in some cases [42].'

The recurrence of nuchal cord in a subsequent pregnancy in the same mother has been reported [43]. Constriction and true knot

recurrence have also been documented by the author although not reported. There have been no known recurrence reports of umbilical cord tumors, cysts or abnormal placental cord insertions. This information is important in order to advise the patient with a UCA loss of future risk of stillbirth.

The following two cases illustrate elements of UCA litigation.

1.1. Case 1

One recent suit in Great Britain (Devine/Mane vs NHS – Scottish Ombudsmen Case #200800763) concerned stillbirth involving hypertension and a nuchal cord. An ultrasound documented a nuchal cord at 23 weeks (Fig. 2). The ultrasound was not repeated. Prenatal visits documented blood pressure changes and weight gain. The patient reported decreased fetal movement. During discovery, NST was noted to contain FHR changes.

Amalia was a full-term healthy baby. Pathology reported asphyxia due to cord occlusion (cord compression). The initial review of the case was in favor of continuing the litigation. The couple ceased litigation in 2013 due to inconsistent specialist obstetric summary reports; both specialists were from the UK and employed within the NHS. The cost of their lawyer was approximately US\$10,000. Compensation was not the driving force of this litigation; this case was purely about the life of baby Amalia and recognition that UCA is indeed a major cause of stillbirth. Prof. Arulkumaran, the then president of the Royal College of Obstetricians and Gynaecologists, held a seminar on UCA due to the Devine case. Since the loss of baby Amalia in 2007, baby Holly Rose Devine was born in Argentina perfectly healthy (personal communication; www.pyramidofantenatalchange.org).

The Devine/Mane case is an example of the challenge presented by the discovery of an umbilical cord anomaly. It is prudent to tell the patient the results of an ultrasound scan and its detail. The images are enhanced and three-dimensional. Finding of an anomaly should follow with a description of the image and instruction to the patient on fetal behavior. Non-stress test (NST) and or biophysical profile (BPP) should be performed as needed. The ethics of this are no different than telling a patient about a positive mammogram. Not informing the patient of a test finding could be viewed negatively in a medico-legal context.

1.2. Case 2

In ongoing litigation from the USA, a 23-year-old gravida 2, para 1 was admitted for decreased fundal size, with



Fig. 2. Gestation scan at 23 weeks. A nuchal cord was present. The parents were not advised. (Reproduced with permission from the parents.)

oligohydramnios noted on an ultrasound and BPP. During admission to labor and delivery, the mother described decreased fetal movement. A BPP was conducted by a junior obstetrician and reported as 10/10. The patient felt that the junior attending was distracted during the study and that there was little fluid noted by the patient. Later, the fetal monitor was taken off the patient in spite of changes in the fetal heart rate, with variable decelerations noted by the patient and a family member. Several hours later, electronic monitoring was used to confirm the FHR during the next nursing shift, and no FHR was found. An ultrasound confirmed stillbirth. There was a nuchal cord, which had been visible on the ultrasound images.

This case illustrates several points. There is a tendency to perform intermittent FHR monitoring in some institutions. This practice may not be advisable if an umbilical cord anomaly is present. The presence of FHR decelerations should not be taken lightly no matter how minimal. In this case, there were obvious variable decelerations recorded prior to stillbirth. If a patient describes decreased movements – especially while hospitalized – the complaint must be taken seriously. Decreased amniotic fluid is a risk factor for umbilical cord compression. A thin umbilical cord exacerbates the problem. A nuchal cord is another risk factor for stillbirth [45].

2. Conclusion

UCA is an important cause of stillbirth. It is now possible to identify pregnancies at increased risk for UCA on ultrasound and potentially manage the compromised fetus. Antenatal FHR monitoring is the best way to detect intermittent umbilical cord compression. If a deceleration to 90 beats/min for 1 min is observed, the observation period may be extended for 24 h to identify additional changes. If fetal behavior or the FHR is abnormal, the observation period may be extended even longer, and, if necessary, delivery should be considered.

These recommendations are based on many peer-reviewed accounts. A few are cited here [46–61]. Further review of UCA can be found at <https://iame.com/online/umbilical/content.php>. Future trends toward consumer 3D imaging/printing will possibly further increase detection of umbilical cord anomalies outside the medical community [62].

Litigation often begins with a misunderstanding and a failure of the medical team to support the family experiencing a stillbirth. UCA-associated stillbirth is a tragic event which is not understood by the family. There is ample research to help explain the reasons behind UCA. It is a fetal event based on fetal reflexes that respond to intrauterine disturbances in blood flow to the fetus. Obstetrical caregivers should be alert to symptoms associated with UCA and be able to evaluate the problem with ultrasound, fetal monitoring, and genetic testing if needed. In the event of a UCA-related stillbirth, disclose all information to the parents. They are usually looking for an explanation, not evidence to initiate litigation. Physicians should welcome every opportunity to answer the patient's questions. What is being sought is reassurance that the cause of death did not result from something the patient did or did not do [62].

Conflict of interest statement

None declared.

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