

Lessons learnt from umbilical cord accidents: are these stillbirths preventable?

Kunaal K Shinde, Vidyadhar B Bangal, Anita Kwatra, Sai K Borawake

ABSTRACT

Umbilical cord accidents (UCA) refer to a group of conditions, where the umbilical cord vessels undergo sudden compression, compromising the blood flow to the fetus. UCA are associated with still birth and are often considered to be an etiology of sudden antenatal death syndrome. Herein, we report three cases of UCA, which reported late to us, resulting in fetal demise.

Key Words: Umbilical cord accidents, true knot, nuchal cord, cord prolapse, stillbirths

Introduction

The umbilical cord connects the fetus to mother through placenta and serves as a lifeline between the two. With an average of length of 55 cm near term [1], it may show variation between 30 through 100 cm. Two arteries and a vein run through it carrying deoxygenated blood away from and oxygenated blood to the fetus respectively. The human umbilical cord is vulnerable to a variety of malformations, lesions, infections, mechanical and iatrogenic events throughout pregnancy, labor and delivery. These conditions make the vessels prone to compression leading to serious deleterious effects on the health of the fetus. Fetal injury or death resulting from compromise to umbilical venous or arterial blood flow is referred to as umbilical cord accidents (UCA) [2,3]. Umbilical cord prolapse, cord entanglement around body parts and true knot formation, are some of the cord accidents responsible for stillbirths as reported in the literature [4]. There are certain obstetric conditions, which predispose cord to meet such accidents, such as cord length abnormalities, malpresentations, premature rupture of membranes, excessive fetal movements and external cephalic version [5,6].

One of the earliest mentions of UCA dates back to 1975 when William Smellie described a case of nuchal cord associated stillbirth in his "Treatise on Midwifery" in 1750, London, England. Andrew Bell published the first drawings of an UCA in the 1st edition of Encyclopedia Britannica (1769) depicting a fetal death with a combination of one nuchal cord, a body loop and a true knot [7]. Herein we report a case series comprising of three cases of UCA from a rural tertiary care hospital in Maharashtra, India.

Case Presentation

Case 1

A 28-year-old Indian woman, gravida 2, para 1 and an unbooked case belonging to low socioeconomic status

Department of Obstetrics and Gynecology, Rural Medical College, Pravara Institute of Medical Sciences, Loni, Maharashtra, India

Corresponding Author

Vidyadhar B Bangal, Email: vbb217@rediffmail.com

presented to our hospital with history of 8 months of amenorrhea. There was no preceding history of vaginal bleeding, discharge, leakage or any loss of fetal movements. Her past medical history was unremarkable and her previous menstrual cycles were regular. She had completed 35 weeks of gestation by menstrual dating method. She had one previous full term normal vaginal delivery. On examination she exhibited pallor with stable vitals and normal respiratory and cardiovascular examination. Abdominal examination revealed a gravid uterus consistent with 30 weeks gestation with cephalic presentation and absent fetal heart sounds. She was not in labor.

Her laboratory investigations revealed hemoglobin of 8.7 gm% and a negative indirect Coomb's test. Total leucocytes and platelet counts were within the normal range. Her blood group was O negative. Serum urea, electrolytes and liver associated enzymes were normal. Liver and renal function tests were within normal limits. Abdominal ultrasound demonstrated a 34 weeks intrauterine dead fetus in cephalic presentation.



Figure 1 True knot in the umbilical cord

The patient was induced with dinoprostone (PGE₂) instillation and prophylactic antibiotics were started. After 10 hours post-induction, patient delivered a still born female weighing 1560 grams with overlapping of fetal skull bones without any obvious anomalies. Fetal Umbilical cord was 70 cm long, thin and ecchymotic. It showed a single tight true knot (Figure 1). Placenta was normal and weighed 250 grams. There were no retro-placental clots or any other

obvious placental pathology. Mother and other relatives were counselled about the cause of intrauterine fetal death and the patient was discharged 3 days after delivery.

Case 2

A 26 year old primigravida at 35 weeks gestation, in labor was referred to our hospital emergently due to dissatisfactory progress of labor. She was a registered case at a private nursing home, and developed labor pains and leaking 12 hr before she was admitted. She noticed decreased fetal movements over the last six hours.

On examination, the patient was hemodynamically stable and is in active labor with good uterine contractions. Her uterine size was consistent with 36 weeks gestation with cephalic presentation. Fetal head was 3/5th palpable. Internal examination revealed mento-anterior face with borderline cephalo-pelvic disproportion. Cardio-respiratory and neurological examination was unremarkable.

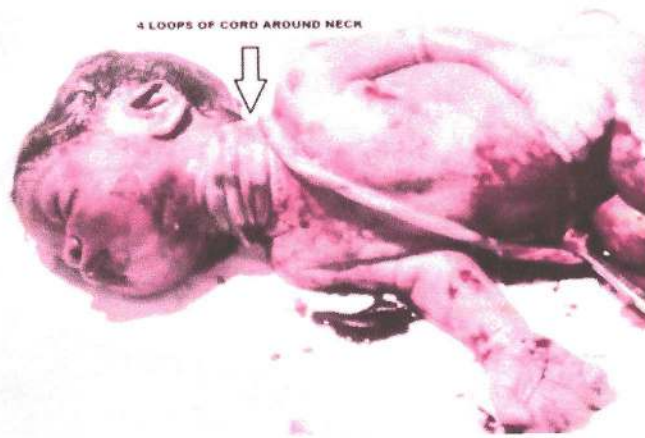


Figure 2 Four loops of cord around neck

Routine hemogram, blood counts, liver and renal function tests were within normal limits. Obstetric ultrasound was not done throughout the period of pregnancy. Due to signs of prolonged labor, patient was posted for emergency caesarean section under spinal anaesthesia. A full term male still-born baby weighing 2.4 kg without any obvious anomaly with 4 tight loops of umbilical cord around neck was delivered (Figure 2). Patient had uneventful post-surgical period and was counselled about the cause of fetal death.

Case 3

A 24 year old, Indian woman, gravida 4, para 3, presented to our hospital at 36 weeks of pregnancy with loss of fetal movements, labor pains and leak per vaginam since ten hours. On examination, her vitals were stable. Abdominal examination revealed a gravid uterus at 34 weeks gestation with horizontally oval contour suggestive of transverse lie with head positioned on right side of the uterus. Lower uterine segment was stretched and fetal heart sounds were absent. Local examination revealed a cervix dilated to 6 cm and a prolapsed umbilical cord outside vagina with absence of cord pulsations (Figure 3). Diagnosis of full-term pregnancy with transverse lie and intra-uterine fetal death with cord prolapse in active labor was made. Obstetric USG confirmed transverse lie and cord prolapse with intra uterine

fetal death. Investigations revealed a Hemoglobin of 7.6 gm% and blood group O +ve. Considering the features of obstructed labor and retracted uterus, an emergency caesarean section was performed and a still-born female of 2.3 kg was delivered. There were no obvious anomalies except features consistent with prematurity. Except for the features of prematurity, there was no obvious abnormality in the baby. Placenta and umbilical cord were normal. Patient was given antibiotics and blood transfusion. Patient and relatives were counselled regarding the cause of death. Post operative period was uneventful and the patient was discharged on the eighth day post-operatively.

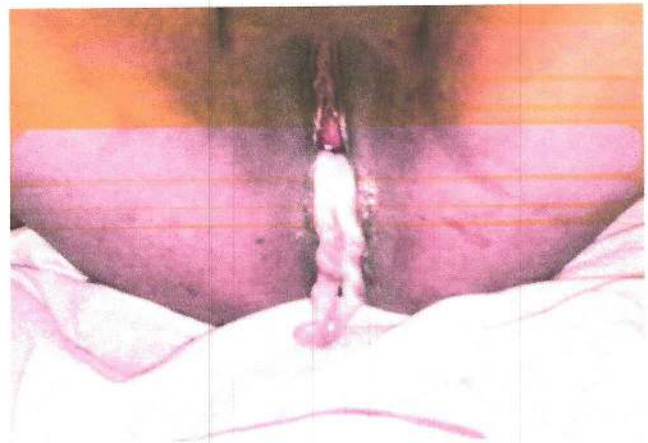


Figure 3 Cord prolapse

Discussion

UCA includes more than 30 conditions which results in compromised blood flow to the fetus [8]. This compromise could prove fatal to the fetus. Stillbirths due to UCA occur generally between 36-38 weeks [9,10]. However UCA as the primary cause of still birth is still debatable. UCA is also one of the reasons of sudden antenatal death syndrome. The exact incidence of UCA is difficult to ascertain, but an analysis of stillbirths by Collaborative Perinatal Project estimates UCAs to have an incidence of 1.5 stillbirths/1000 births [8]. Among the various manifestations of UCA, we discuss herein literature relevant to the cases reported.

An umbilical cord knot may result in UCA as seen in our first case. About 1% of babies are born with one or more knots in the umbilical cord [11]. A loose knot may not compromise the blood flow, whereas a tightening knot during descent could potentially occlude the circulation. The knot can be a true knot or a pseudo knot. A true knot is an actual knot in the umbilical cord while a pseudo knot is formed by the twisting of the umbilical vein around the artery resulting in the localized thickening of Wharton's jelly. The conditions that predispose to a true knot include long cord (>80 cm), polyhydromnios, small size of fetus or multiple fetuses [12]. On the contrary, in our case the cord was only 70 cm long. Srinivasan et. al. reported 4 true knots in a case of live birth with long umbilical cord (127cm) [13]. However, the presence of a true cord increases the risk of fetal demise by several folds [14]. In less severe form, knots have been implicated as the cause of intrauterine growth retardation [13,15]. Prenatal diagnosis is difficult with limited help of Doppler flow study.

Multiple tight loops of cord around the neck (nuchal cord) can result into acute placental insufficiency and even strangulation of the fetus. Nuchal cord can be of two forms: one with 360 degrees loop around the neck with the placental end crossing over the umbilical end while the other form where the crossing over takes place under the umbilical end. In the former the neck gets entangled in an unlocked pattern that can undo itself while in the latter undoing is not possible. Unlike our case, still birth is not very common and this form of UCA is associated with better prognosis [16]. Nuchal cord is associated with variable fetal heart rate [17], decreased fetal movement and in extreme conditions, intrauterine fetal demise. Excessive fetal movement or long umbilical cords are risk factors [18]. Nuchal position of cord or cord entanglement can be diagnosed easily by careful 4D ultrasonography and color doppler studies [19-20].

Prolapse of umbilical cord occurs in about 1 in 300 births [11]. It is an obstetric emergency that occurs either late during pregnancy or in labor. The risk factors include breech, preterm labor, long cord, polyhydromnios, premature rupture of membrane and twins. It is the second twin which is more commonly affected. Weekly transvaginal sonography after 36 weeks can result in early prediction and prevention of cord prolapse [21]. Fetal outcome is directly related to the time lapse between diagnosis and effective management [22-23]. Continuous monitoring of fetal heart rate is vital to make prompt diagnosis of cord prolapse during labor. However absence of pulsation is not a very reliable indicator of prolapse. Once detected, immediate measures to relieve the pressure on cord could prevent fetal distress and further complications. Some of these rapid measures include manual elevation of the presenting part and placing the patient in the Trendelenburg position. The rate of Cesarean section in cord prolapse has increased, improving the fetal outcome. Katz et. al. report in a five year study of 57 cases of cord prolapse, the initial management by filling the bladder with 500-700 mL of saline and by intravenous ritodrine followed by Cesarean section at the earliest. [24]. Ante-natal diagnosis of cord prolapse is challenging because it is difficult to predict. Repeated sonography in late phase of pregnancy and strict vigilance during labor should be followed.

UCA can occur at any period of gestation from conception to delivery, with higher risk in the third trimester. Other commonly encountered UCA include vasa previa, marginal insertion, monoamniotic twins and body loop. A discussion of all forms of UCA is beyond the scope of this article. It is a highly unpredictable complication of pregnancy and its association with sudden antenatal death syndrome makes it an intensely debated topic. The risk factors for UCA are often non-modifiable and early diagnosis being difficult. As seen in our case series, ante natal diagnosis could only be made in one case.

Conclusion

Stillbirths due to UCA can be reduced by identifying risk factors. All the forms of UCA are not associated with fatal outcome. The role of investigating modalities like sonography and color Doppler along with quick action to minimize the time interval between diagnosis and treatment will help to keep the rate of stillbirths in UCA at its lowest.

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Consent

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the editor-in-chief of this journal.

Authors' Contributions

KKS, VBB, AK participated in the clinical diagnosis, sequence alignment, drafting the manuscript and made useful contribution in the review of the literature. VBB, KKS and SKB were the operating team of surgeons. VBB and SKB participated in writing discussion. KKS, VBB and AK helped in the revision of the manuscript. All authors read and approved the final manuscript.

Competing Interests

The authors declare that they have no competing interests.

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