TWISTING OF THE UMBILICAL CORD CAUSING INTRAUTERINE FETAL DEATH

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Abstract

Umbilical cord accident (UCA) is an emergency situation, as it threatens fetal wellbeing and or results into fetal jeopardy. Cord prolapse, cord compression, cord entanglement, true knot formation, thrombosis and rupture of cord blood vessels are some of the known cord related causes of stillbirths. Twisting of the umbilical cord occurs less often than above mentioned cord accidents. Twisting of the umbilical cord leads to obstruction in the flow of blood in umbilical cord vessels leading to acute placental insufficiency and sudden fetal death. A rare case of full term pregnancy with multiple twists of the umbilical cord, leading to intrauterine fetal death is presented in this case report. The antenatal diagnosis, predisposing factors and preventive measures are discussed in the end.

Keywords: Twisting of umbilical cord, Intrauterine fetal death, Umbilical cord accidents, Stillbirths

1. Introduction

Umbilical Cord Accidents (UCA) are responsible for sudden antenatal death syndrome (SADS). Meticulously performed ultrasound in prenatal period can identify most of the umbilical cord accident. An Umbilical Cord Accident (UCA) occurs, when umbilical venous or arterial blood flow is compromised to a degree that it leads to fetal injury or death.1,2 The human umbilical cord is vulnerable to a variety of malformations, lesions, infections, mechanical and iatrogenic events throughout pregnancy, labor and delivery. Medical literature on UCA has accumulated since the Collaborative Perinatal Project (CPP).3,4,5 Two reviews of UCA from the CPP study including; multiple twists, true knot, nuchal coils, and body loops suggests an association with extremes of umbilical cord length as a fetal risk factor for fetal malformation, injury and stillbirth.6,7 A review of stillbirths from the CPP suggests that UCA’s have at least an incidence of 1.5 stillbirths/1000 births.8

2. Case report

Twenty three year old, unbooked second gravida presented with history of nine months of amenorrhoea with loss of fetal movements since five days of admission. She had undergone emergency caesarean section in her first pregnancy for cephalo pelvic disproportion three years back in private hospital. Her intra and post operative period following caesarean section was uneventful. In the present pregnancy, she had undergone anomaly scan at 18 weeks of gestation. There was no history of leaking or bleeding per vaginum in present pregnancy. She had only two antenatal visits in the present pregnancy. She did not receive hematinic tablets. She did not suffer from any major medical or surgical illness in the past. On examination, she had moderate degree anaemia. Her other vital parameters were within normal limits. Per abdominal examination revealed full term gestation with fetus in transverse lie. Fetal heart was absent. Uterus was relaxed and liquor volume was less. Estimated weight of the baby was three kilograms. There was no scar tenderness and the abdominal scar was healthy. Pelvic assessment revealed contracted pelvis. Laboratory investigations showed evidence of iron deficiency anaemia (Hb-6.9 grams percent). Other hematological and biochemical investigations were within normal limits. Obstetric ultrasound showed single intrauterine dead fetus in transverse lie of 36 weeks gestation. Expected fetal weight was 2.9 kilograms. Amniotic fluid index was 3 centimeters. Placenta was situated at fundus and there was no demonstrable fetal, placental or cord abnormality. She was transfused with two pints of fresh whole blood. She underwent elective caesarean section under general anesthesia. A full term dead, male baby weighing 3.1 kilograms was delivered by caesarean section. Placenta and cord was delivered. There were no fetal abnormalities except for the early evidence of maceration in the form of skin peeling. Placenta was normal in morphology. There was no evidence of abruption or gross calcification. Umbilical cord was 55 centimeters long. It was edematous,
congested and had three twists at its fetal attachment near umbilicus. (Photograph 1 and 2) The twisted portion of the cord was pale white in color and had thinned out. Clinical autopsy of the baby could not be performed due to lack of parental consent. The post operative period was uneventful. She was discharged on tenth postoperative day. She was given dietary and contraceptive advice and was asked to come for postnatal visit. She along with her relatives was counseled about the cause of stillbirth and the precautions to be taken in future pregnancies.

3. Discussion
The umbilical cord serves as a critical lifeline to the developing fetus. The umbilical cord is the principal connection between the fetus and the placenta, providing the nutrients, oxygen and fluids necessary for life in utero. On occasion, a variety of intrinsic or accidental processes involving the umbilical cord can result in intrauterine fetal death. The umbilical cord is vulnerable to a number of insults which may alter cord morphology, diminish cord blood flow and ultimately compromise fetal nutrition. The cord and its constituent tissues, an outer layer of amnion, porous Wharton’s jelly, two arteries and one vein, are designed to provide and maintain the blood flow to the developing fetus. Some of the morphological aspects of the umbilical cord, such as its length, knots, insertion to the placenta, number of vessels and twisting have been associated with pathological outcomes. At term, the typical umbilical cord length is 55 to 60 cm. Adverse outcomes have been reported with both abnormally long (70 to 80 cm) and abnormally short cords (30 to 40 cm). Long or short cords can be the cause of hematomas and thrombosis of cord vessels and the surface of the placenta thus causing fetal hypoxia, damage of the central nervous system or even fetal death.

Umbilical cord torsion has been reported to be an uncommon cause of intrauterine fetal demise. Although initially described more than 300 years ago, relatively few cases have been described in the literature in the last 50 years. It may result from fetal movement during which the cord normally become twisted. But recent reports have shown familial clustering. Such intra familial clustering suggests that a genetic predisposition for umbilical cord torsion may exist. Umbilical cord torsion has usually been regarded as secondary to fetal death or cord constriction or due to lack or abnormality of Wharton’s jelly. Umbilical cord torsion, in the absence of predisposing constriction or abnormality of Wharton’s jelly, can obstruct the umbilical blood vessels and cause intrauterine death. Prenatal ultrasonography can recognize torsion of the umbilical cord. If the vein-to-vein pitch is <2 cm, torsion may be associated. Cardiac failure can occur with umbilical cord torsion and can present as nonimmune hydrops. Results of the studies suggest that non genetic factors affect the twisting of the cord, with shared and non-shared environmental factors explaining most of the variation. Previous research has debated over whether twisting is genetically determined, inherent to the cord itself or the result of external/extrinsic forces. Fletcher reported about a mechanical rotation in which the cord twists as a result of fetal movement or rotation in early gestation. On the other hand, alternative theories have suggested that twisting is the result of factors inherent to the cord itself. Malpas and Symonds have suggested that the helical structure of the cord results from genetic differences in the direction of the fibres in vessel walls and that a reciprocal action between the vessels walls and flow rate of the fetal blood result in the umbilical cord twist. Cord twisting may occur either 1) clockwise, 2) mixed/undefined and 3) counter-clockwise. The literature on umbilical cord knots and twisting suggests that more adverse outcomes are associated with real knots or clockwise twisting, while the frequency of problems diminish in the other categories.

In the present case, woman experienced loss of fetal movements 8 days before coming to the hospital. Due to lack of awareness regarding need for reporting this symptom and for many other reasons, she continued to stay at home. Fetal demise was diagnosed during her subsequent antenatal visit. There was no evidence of growth restriction. As she was a case of previous cesarean section with full term baby in transverse position with oligohydramnios, external cephalic version was not attempted. The cause of fetal demise was thought to be related to umbilical cord. Twisting of the cord was not diagnosed by ultrasound. The cause was confirmed after the delivery of the baby by elective caesarean section.

Conclusion
Twisting of the umbilical cord is a rare but important cause of intrauterine death. The
torsion or the twist can be diagnosed during antenatal period by meticulously performed obstetric sonography. Woman complaining of either increased or decreased fetal movements or with growth restriction must be carefully evaluated for cord related causes of fetal distress. Sonologist must make an attempt to visualize entire length of cord and should rule out cord related conditions which are responsible for fetal jeopardy.

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CLINICAL PHOTOGRAPHS OF TWISTING OF UMBILICAL CORD