Case Report

Prenatal diagnosis of single umbilical artery complicated by intrauterine growth retardation and preterm labor: Case report

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Abstract

Fetuses with single umbilical artery (SUA) at great risk of intrauterine growth retardation (IUGR), intrauterine fetal death (IUFD) and prematurity. A 24-years-old woman, 28 weeks` gestation, presented to the Ahmadi hospital, Kuwait, with history of preterm premature rupture of fetal membranes (PPROM). After exclusion of the PPROM, the ultrasound scan of the studied woman showed; asymmetrical IUGR with SUA. The diagnosis of SUA confirmed by the color flow Doppler. She delivered spontaneously at 36 weeks+2, and a cut section in the umbilical cord done to confirm the diagnosis of SUA. The congenital and chromosomal abnormalities of the studied neonate excluded after normal pelvi-abdominal, brain ultrasound and normal karyotyping (46, xx); respectively. The prenatal diagnosed SUA in the studied cases associated with IUGR, preterm labor (PTL) and small for gestational age (SGA). SUA can be considered a marker of diagnosable congenital fetal malformation (CFM) and aneuploidy.

Keywords: Artery, intrauterine growth retardation, preterm, single, umbilical

Introduction

The umbilical cord normally contains one umbilical vein and two umbilical arteries. The umbilical vein carries the oxygenated blood to the fetus from the placenta and the arteries carry the deoxygenated blood from the fetus to the placenta.[1]

Single umbilical artery (SUA) may be due to primary agenesis or secondary atrophy of one of the umbilical arteries.[1]

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Secondary atrophy of one of the umbilical arteries can be seen due to multiple pregnancies, advanced maternal age, smoking, medical disorders with pregnancy as diabetes, hypertension and medications intake with pregnancy (phenytoin, levothyroxine, and vitamin A), and placental abnormalities.[2]

Primary agenesis of one of the umbilical arteries is commonly associated with chromosomal abnormalities and congenital fetal malformations (CFM).[2]

Fetuses with SUA are at great risk of intrauterine growth retardation (IUGR), intrauterine fetal death (IUFD), prematurity and low birth weight (LBW).[1,2]

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Case Report

A 24-years-old pregnant woman, gravida 2 (P0 + 1 (previous first trimester spontaneous miscarriage)), 28 weeks` gestation according to her last menstrual period (LMP) with past history of laparoscopic gastric sleeve two years ago, presented to the emergency room with history of gush of water (query preterm premature rupture of fetal membranes (PPROM)).[3-6]

The PPROM excluded after speculum examination, nitrazine test, AmnioQuick Duo test and amniotic fluid index (AFI).[3-7]

The ultrasound scan of the studied woman showed decreased fetal abdominal circumference by two weeks than the calculated gestational age according to her LMP with normal biparietal diameter, head circumference, and femur length indicating asymmetrical or type 1 (head spring) IUGR with SUA.[8]

Doppler ultrasound studies showed normal umbilical blood flow and the color flow mapping confirmed the diagnosis of SUA [Figure 1].

Strict antenatal follow up according to the hospital protocol including weekly twice cardiotocography (CTG), weekly umbilical artery Doppler, and ultrasound for fetal growth and fetal biometry every two weeks till delivery.[9]

She delivered spontaneously at 36 weeks $^{+2}$ gestation, a baby girl 1.910 Kg small for gestational age (SGA) with APGAR score 7, 8, 8 at 1, 5, and 10 minutes; respectively.

After separation of the placenta, the placenta was carefully examined, and the placental abnormalities excluded. A cut section in the umbilical cord was done to examine the umbilical cord vessels [Figures 2 and 3] and sent for microscopic examination to confirm the diagnosis of SUA.

General neonatal examination by the senior neonatologist was completely normal and neonatal echocardiography showed normal findings for the premature babies (small patent 3-4 mm foramen ovale (PFO)). When the pelviabdominal and the brain ultrasounds of the studied neonate were normal and the karyotyping of the studied neonate confirmed normal karyotyping (46, xx), the studied neonate was discharged for follow-up in the outpatient department. An ethical committee approval and a written consent from the studied woman were taken to publish her data as a case report.

Discussion

The incidence of SUA varies from 0.2 to 0.87%.[10] Prenatal ultrasound evaluation for SUA should be done during the second and third trimesters of pregnancy.[10,11]

The presented case was diagnosed with SUA and asymmetrical or type 1 IUGR by prenatal ultrasound at 28 weeks` gestation. The patient delivered spontaneously preterm delivery at 36 weeks^{+2} gestation. The prenatal diagnosis of asymmetrical or type 1 IUGR confirmed after delivery of SGA neonate (1.910 Kg at

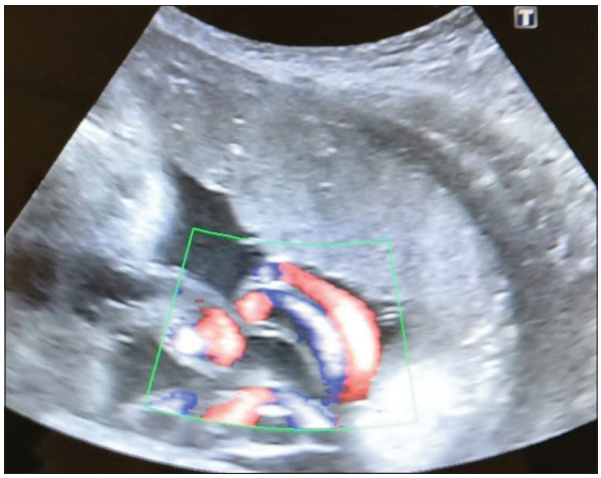
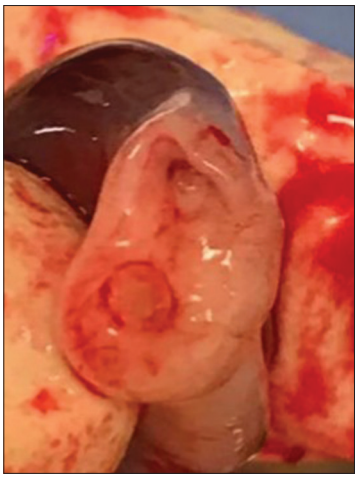
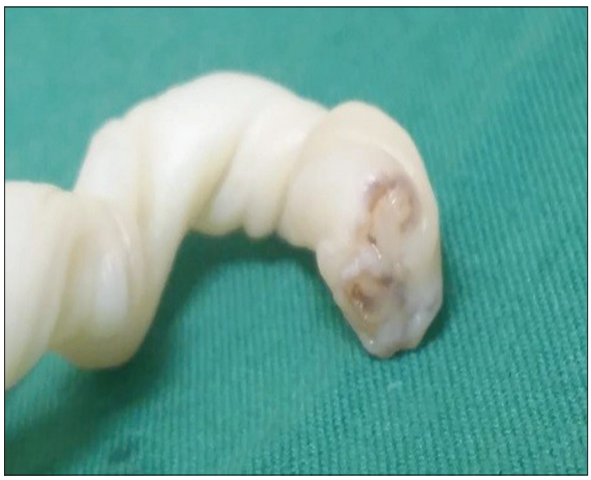


Figure 1: Single umbilical artery confirmed by color flow mapping



Figures 2: Cut section of the umbilical cord immediately after delivery of placenta showing single umbilical artery



Figures 3: Cut section of the umbilical cord 2 hours after delivery of placenta showing single umbilical artery

36 weeks^{+2} gestation). In addition, the prenatal diagnosis of SUA was confirmed by a cut section of the umbilical cord and by the microscopic examination of the cut section of the cord.

IUGR defined when the rate of fetal growth is less than normal growth potential of a specific infant as per the race and gender of the fetus.[10] Asymmetrical (type I) or "head sparing" IUGR occurs later in third trimester and caused by utero-placental insufficiency.[10,11] Ultrasound parameters of asymmetrical or type 1 IUGR shows decreased abdominal circumference with normal biparietal diameter, head circumference and femur length.[10,11]

SGA diagnosed when the birth weight is less than 10th percentile for that gestational age for the population norms on the growth charts.[10,11]

Authors reported that fetuses with SUA at high risk for adverse pregnancy outcome as IUGR, IUFD, prematurity and LBW.[10,11]

Neonates with SUA are at risk of CFM and chromosomal abnormalities and the commonest CFM associated with SUA are renal, cardiovascular and musculoskeletal malformations.[1]

Neonates with SUA have 15 times higher risk of chromosomal abnormalities and a significant proportion of infants with SUA may have isolated occult renal anomalies.[1-10]

The congenital and chromosomal abnormalities of the studied neonate excluded after normal pelvi-abdominal, brain ultrasounds and normal karyotyping (46, xx); respectively done on the next day following delivery.

SUA can be seen as isolated finding or associated with congenital heart disease (CHD).[10-11] The studied neonate CHD excluded by echocardiography done on the next day following delivery which showed normal findings for the premature babies (small patent 3-4 mm PFO).

Murphy-Kaulbeck *et al.*, found that the SUA fetuses and neonates had 6.77- and 15.35-times greater risk of CFM and chromosomal abnormalities; respectively.[2]

Murphy-Kaulbeck *et al.*, found the commonest fetal malformations in chromosomally normal fetuses and neonates with SUA were genitourinary (6.48%), cardiovascular (6.25%) and musculoskeletal (5.44%) and concluded that the detection of SUA is important for prenatal diagnosis of CFM and aneuploidy.[2]

The Society of Obstetricians and Gynaecologists of Canada recommend a detailed review of fetal anatomy with SUA and follow-up assessment of fetal growth.[1,2] Parents should be counseled regarding the increased risk for poor outcome, repeated ultrasound examination, Doppler assessment and testing for fetal well-being in late weeks of pregnancy.[2]

SUA considered a marker of diagnosable CFM and it is recommended to assess the number of cord vessels during the 12th week ultrasound scan.[1,2]

Conclusion

The prenatal diagnosed SUA in the studied cases are associated with IUGR, PTL, and SGA. It is useful to evaluate the umbilical cord vessels during pregnancy to improve the pregnancy outcomes as SUA can be considered a marker of diagnosable CFM and aneuploidy.

Authors' contribution

IAA is responsible for study design, ultrasound, follow-up of the studied cases and submission for this case report publication. MAF was responsible for Microsoft editing, update of references, and final revision before publication. MESH was responsible for ultrasound, Microsoft edit, and final revision before publication. OOA was responsible for intellectual content and final revision before publication.

SS is responsible for intellectual content and update of references. GZ is responsible for Microsoft editing, update of references, and final revision before publication.

Declaration of parents' consent

The authors certify that they have obtained all appropriate consent forms from the parents. Parents have given their consent to use their neonate images and other clinical information to be reported in the journal. Parents understand that their neonate name and initials will not be published, and due efforts will be made to conceal the identity, but anonymity cannot be guaranteed.

Compliance with Ethical Standards

Financial support and sponsorship

The case report was funded by the authors themselves.

Conflicts of interest

There are no conflicts of interest.

Research involving Human Participants

A departmental approval taken, and an informed written consent taken from the studied woman to publish her data for scientific activity and as a case report.

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