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Case report

Twin to Twin Transfusion Syndrome

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Abstract

Introduction: *Collateral arterial growth is an effective adaptation process to maintain blood supply in myocardial tissue distal to a coronary stenosis and in other vascular beds where feeding arteries are obstructed (1) . The drive for outgrowth and remodelling of arterial collaterals is an increased shear stress due to an enlarged blood flow occurring after the event of stenosis in a near by conduit artery. Remodelling includes increased growth of the diameter and length of the collateral, causing the characteristic tortuous or cork screw appearance (1) .Whilst collateral growth is generally beneficial, we will present evidence that arterial collateral out growth occurred in a monochorionic twin placenta where it jeopardised the pregnancy by causing the twin to twin transfusion syndrome (TTTS).*

Objective: *to report a case of twin-to-twin transfusion*

Case Report: *Reporting a case experienced and taken care in our Clinic Fetomaternal RS. Dr .M. Jamil Hospital. A 34 years old woman, gravida 2, para1, at 31 weeks and 6 days amenorrhea, because her pregnant uterus was too large for gestational Referral to tertiary center followed. At 32 weeks and 2 days amenorrhea biometry indicated estimated fetal weights of 1950 and 1450 g (Hadlock), oligohydramnios and polyhydramnios (amniotic fluid index:23cm. No structural anomaly of the heart was seen. No interventions, one course of corticosteroids were administered for fetal lung maturation. At 33 weeks a slight decrease in heart rate variability of the smaller twin was observed. A Caesarean section under spinal anesthesia was performed. Two girls were delivered of 1585 g and 2135 g and in good condition (Apgar scores 8 and 9 after 1 and 5 min in both girls). The monochorionic diamniotic placenta weighed 740 g. The cord of the recipient was centrally inserted and that of the donor velamentally. Placenta was born monochorion, diamnion.*

Conclusion*: TTTS is caused due to unidirectional deep arteriovenous (AV) anastomoses with the superficial short comings. Hypovolemia, oliguria and oligohydramnion occurs in the donor fetus. Hypervolemia, polyuria and polyhydramnios occurs in the recipient fetus*

Keywords: *twin to twin transfusion syndrome, ultrasonography, monochorion*

INTRODUCTION

Collateral arterial growth is an effective adaptation process to maintain blood supply in myocardial tissue distal to a coronary stenosis and in other vascular beds where feeding arteries are obstructed.[1] The drive for outgrowth and remodelling of arterial collaterals is an increased shear stress due to an enlarged blood flow occurring after the event of stenosis in a near by conduit artery. Remodelling includes increased growth of the diameter and length of the collateral, causing the characteristic tortuous or cork screw appearance.[1] Whilst collateral

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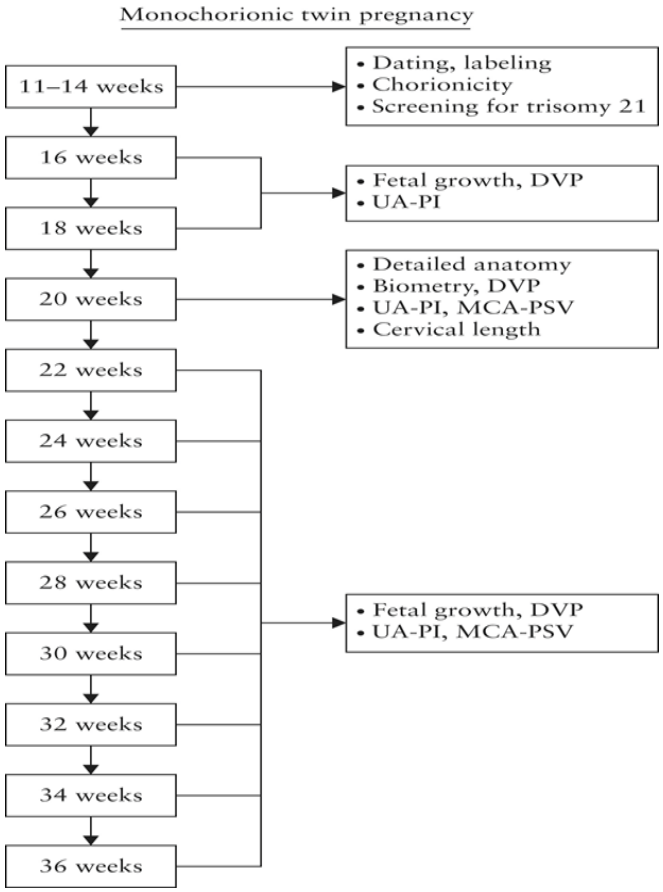

ISUOG Practice Guidelines: role of ultrasound in twin pregnancy

CASE REPORT

A 34 year old woman, gravida 2, para1, was seen in her local hospital at 31 weeks and 6 days amenorrhoea, because her pregnant uterus was too large for gestational age. Ultrasound examination revealed twins, and no intertwin membrane was seen. Al ready a slight difference in foetal size was observed. Repeat ultrasonography one week later showed a very thin inter twin membrane, one placental mass, no lamda sign. Detailed scanning showed an increased echogenicity of the intestines, raising suspicion of a bowel occlusion in the smaller twin. The obvious difference in biometry and a considerable difference in amniotic fluid compartments of the twins raised suspicion of TTTS. Referral to Fetomaternal RS. Dr .M. Jamil Hospital of tertiary centre followed. At 32 weeks and 2 days amenorrhoea biometry indicated estimated foetal weights of 1950 and 1450 g (Hadlock), oligohydramnios and polyhydramnios (amniotic fluid index:23 cm). No structural anomaly of the heart was seen. The larger twin had an enlarged, fully filled bladder during the complete examination. The smaller twin had increased echogenicity of the bowel compartment, but no abnormal dilatations were seen. Some bladder filling was observed. When one week later the discrepancy in fluid compartments further

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increased (AFI:26 cm), further deterioration to a full blown oligohydramnios/ polyhydramnios sequence with one twin stuck was feared. No preterm contractions and no discomfort arose from the polyhydramnios. No interventions, such as puncture of the intertwin membrane, amniocentesis or laser therapy were performed. One course of corticosteroids were administered for foetal lung maturation. At 33 weeks a slight decrease in heart rate variability of the smaller twin was observed. A Caesarean section under spinal anaesthesia was performed. Figure 2. two girls were delivered of 1585 g and 2135 g and in good condition (Apgar scores 8 and 9 after 1 and 5 min in both girls). The monochorial diamniotic placenta weighed 740 g. The cord of the recipient was centrally inserted and that of the donor velamentally. By inspecting the cotyledon are masses supplied by each chorionic vasculature, it was estimated, that there was a 3:1 unequal sharing of the placental mass (recipient : donor). After one day both twins were transferred to the neonatal ward of hospital. Follow up of both children at the child department of the Dr.M.jamil Hospital shows no developmental problems in either of the children to date (3months old).

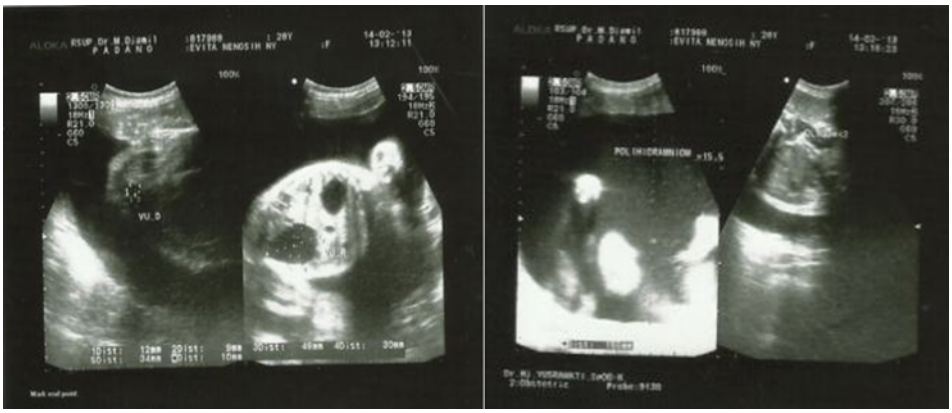

Figure 1. Fetal bladder in donor twin is empty and fetal bladder in recipient twin is over distency, Polyhidramnion: excess of amniotic fluid.

Figure 2. Baby in case 2 with Discordance Twin : difference in size greater than 10% in 1585 g and 2135 weight at birth.

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DISCUSSION

We propose that it should be possible to predict the underlying placental anatomy by frequent detailed ultrasound scanning including full biometry, as soon as monochorionici ty is established. Collecting consecutive cases of monochorionic twins using serially ultrasound monitoring and with definite pathological studies of the placentae is desperately needed for antepartum classification of monochorionic twins according to their placental problem. Early serial ultrasound scanning including full biometry, constructing growth curves with difference average ratio plots could make early recognition possible. This serial ultrasonography should also include assessments of the foetal hearts. Differences in heart/thoracic ratios or appearance of tricuspid regurgitation can be considered evidence for the haemodynamic effects of foeto foetal transfusion.[2] Also differences in foetal bladder filling and in amniotic fluid compartments should be looked for. The ratio of the pulsatility indices of the umbilical arteries may serve as an indicative measure of placental sharing, as the umbilical artery pulsatility index reflects placental resistance, which is proportional to placental mass. From these sonographic data, the various patterns predicting the underlying placental problem may ensure better diagnostic antepartum criteria. Hopefully this type of pattern recognition will lead to early identification of cases requiring treatment. We propose that an expert team comprised of an experienced ultrasonographer, a dedicated obstetrician, a neonatal intensive care specialist, a pathologist experienced in developmental pathology, and a developmental neurologist should manage monochorionic twin pregnancies.

Monochorionic twin pregnancies constitute about 75% of monozygotic (identical) twins and share one common (monochorionic) placenta shows Figure 3. These pregnancies are considered to be at high risk for adverse outcome.[3] predominantly because about 15% of them develop TTTS, a severe complication of monochorionic twinning with a high incidence of intrauterine mortality and premature birth. TTTS diagnosis is by a midtrimester discordance in amniotic fluid volumes, i.e. the oligo polyhydramnios sequence (donor versus recipient), with signs of hypovolemia, hypotension and growth restriction in the donor, and hypervolemia, hypertension and cardiac dysfunction in the recipient shows.

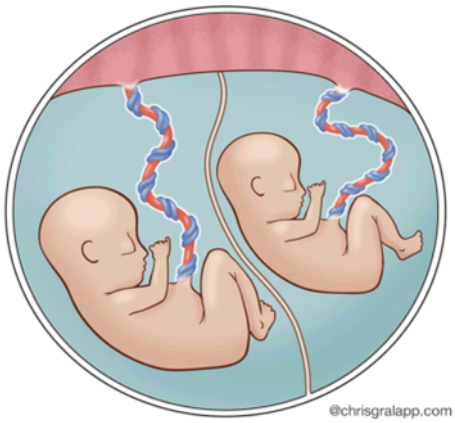

Figure 3. Normal monochorionic twin

Virtually all monochorionic twin placentas have vascular connections that link the two fetoplacental circulations. In arteriovenous anastomoses, a joint placental cotyledon is supplied

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by arterial blood from one twin (the donor) and drained by venous blood to the other twin (the recipient). Arterio arterial anastomoses directly connect the arterial circulations and venovenous anastomoses the venous circulations of the twins. TTTS may result from arteriovenous anastomoses, which cause a continuous increasing transfusion of blood from the donor to the recipient.[4] Arterio arterial and veno venous anastomoses connect the hypertensive recipient with the hypo tensive donor. These anastomoses reduce the primary arterio venous inter twin transfusion, and thus mitigate the severity of TTTS, or even prevent it. Support for the protective role of arterio arterial anastomoses comes from theoretical analysis and from a TTTS case where late onset at 34 weeks of gestation occurred.[5] Shows Figure 4.

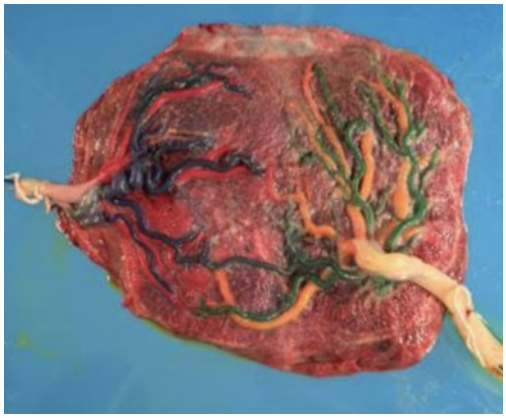

Figure 4. Monochorionic placenta with the vasculature of twins in blue/red and in green/yellow (Courtesy of Anthony Johnson)

TTTS developing in cases of TTTS that lack arterio venous but contain arterio arterial anastomoses includes a stenosed recipient chorionic artery that connects with the arterio arterial anastomosis. After onset of the stenosis the consequential reduced arterial pressure at the recipient side of the anastomosis causes an increasing inter twin arterio arterial transfusion, which causes TTTS, but also an increased shear stress in the vascular wall of the anastomosis, triggering the development of the anastomosis as a tortuous collateral artery.[6]

The twin to twin transfusion syndrome (TTTS) is a serious complication of monochorionic twins with considerable perinatal morbidity and mortality.[6,7] As a consequence, antenatal treatment is surrounded by controversy. The basis of the syndrome is a placental vascular anomaly.[8] Placental anastomoses linking the two foetoplacental circulations produce an uncompensated net transfusion of blood from the donor to the recipient twin. Remarkably, however, the placenta does not seem to play a major role in therapeutic management decisions, and is rarely adequately investigated following delivery. Insufficient data are available relating diagnosis, foetal growth and therapy with placental anastomotic patterns.

We present a case of a monochorionic twin pregnancy showing serial ul trasonography obtained biometry, clinical and placental followup, and evaluation of the data by a haemodynamic.

The arterio arterial anastomosis in the TTTS case represented a functional collateral artery, whose out growth was driven by an increased shear stress caused by an increased flow to a lower pressure vascular bed distal to the stenosed recipient chorionic artery. The rationale

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for the TTTS is that once the stenosis developed, it created a substantial drop in arterial blood pressure distal as compared to proximal of the stenosis, considered from the recipient's direction of flow. This decreased pressure extended to the recipient side of the arterio arterial anastomosis. The resulting pressure difference that now existed between donor and recipient sides of the anastomosis led to an increasing net intertwin transfusion through the arterio arterial anastomosis (donor to recipient). The associated increased wall shear stress drove the arterio arterial anastomosis to become a functional collateral artery, stimulating increased growth of its diameter and length, causing the cork screw appearance. As a consequence, the donor twin not only maintained blood supply to the lower pressure recipient placental bed distal to the stenosis, i.e. the beneficial function of collaterals, but at the same time also created a continuous increasing loss of its blood volume and corresponding gain of blood volume for the recipient, which is the condition for onset of TTTS.[9] In conclusion, although arterial collateralisation normally protects the distal vascular bed from becoming deprived of blood supply, we believe to have shown strong evidence that an existing arterioarterial anastomosis which connected to a chorionic artery that developed a stenosis remodelled to a functional collateral artery with tortuous appearance.

CONCLUSION

TTTS is caused due to unidirectional deep arteriovenous (AV) anastomoses with the superficial short comings. Hypovolemia, oliguria and oligohydramnion occurs in the donor fetus. Hypervolemia, polyuria and polyhydramnios occurs in the recipient fetus.

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