Twin To Twin Transfusion Endangering The Mother

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Abstracts: There was a case of Twin to twin transfusion syndrome (T.T.T.S.) with one dead monster baby and other alive low birth weight baby, leading to early Disseminated Intravascular Coagulation(D.I.C) in mother, in Obstetric ward of C.R. Gardi Hospital Ujjain, (M.P) India. [Saluja J K NJIRM 2014; 5(2) :139-141]

Key Words: Twin to Twin Transfusion, Fetal transfusion syndrome, Fetal-Fetal Transfusion Syndrome

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Introduction Twin to twin transfusion syndrome (T.T.T.S.) was first described by a German Obstetrician Friedrich Schatz in 1875[1]. Twin to twin transfusion syndrome is observed in 10-15% Monochorionic twin pregnancies. Alternative names are Fetal transfusion syndrome, Fetal-Fetal Transfusion Syndrome (.F.F.T.S). It complicates approximately 1 in 5 of all monochorionic diamniotic twin pregnancies[2]. The pathogenesis is still unknown and the mortality reaches 80-90% if not treated[3]. Malformation in monozygotic twin is 2.1% as compared to incidence of 1% in singleton pregnancy[4]. Twin to Twin syndrome is a rare condition that occurs only in identical twins when they are in the womb. In this blood is transfused from a donor twin to the recipient sibling. The donor becomes anemic and the growth may be restricted, whereas the recipient becomes polycythaemic, may develop circulatory overload and manifested as hydrops.

Criteria for Twin to Twin Transfusion syndrome (Bruner and Rosemond)[5] : (a) Same sex fetus, (b) Monochorionicty with placental vascular anastomosis, (c) Weight difference between twins greater than 20%, (d) Hydramnios in large twins and oligohydramnios in smaller twin, (e) Hemoglobin difference greater than 5gm /dl, as a result of single placenta the blood supply of monochorionic twin fetuses can become connected. Although each fetus uses its own portion of placenta the connecting blood vessels within the placenta allow blood to pass from one twin to the other. Disproportionate transfer of blood from one twin (donor) to the other (recipient) and anastomosis on the chorionic plate are responsible for net imbalance of blood flow and subsequent development of T.T.S. All this can be diagnosed ultrasonographically in antenatal period except Hemoglobin percentage.

Case Report: A 23 yrs old second gravida came as an emergency on with full term pregnancy with labor pains with an ultrasound report and Colour Doppler study reports of two weeks before documenting twin pregnancy with one live fetus, and other malformed dead fetus. On general examination nothing specific, abdominal examination: distended &tense uterus with multiple fetal parts and first baby vertex presentation. FHS 150/min regular. Vaginal examination:- Cervix 2-3 cm dilated, 90% effaced, head at ischial spine. Investigations within normal limits.

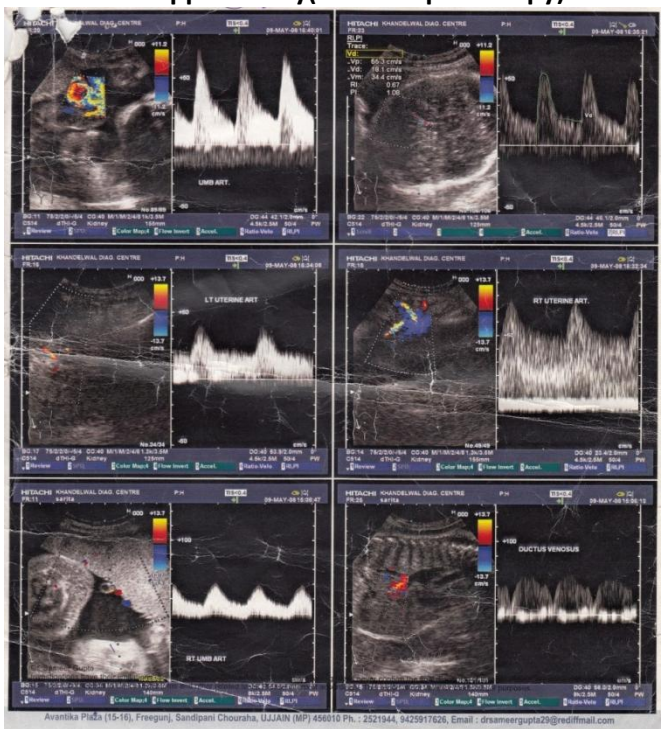
Ultrasonography Reports : Fetus A : Normal fetus 35wks pregnancy. Cephalic FHR 151/minute, liquor adequate, AFI-15 cm. Fetus B : A Malformed fetus with absent head and swollen abdomen and edematous skin on the right side. No cardiac activity seen.A separate 3 V umbilical cord inserting into the placenta cell.

Impression Twin pregnancy with single live fetus , separating membrane present, placenta – single and anterior.Grade II

Color Doppler Study Shows :LEFT SIDE – Umbilical artery shows normal pulsatility index and waveforms.No reversal seen. Middle cerebral artery shows normal pulsatility index and waveforms. No evidence of redistribution seen. Bilateral uterine arteries are normal in waveform. No notching seen. The fetal heart appears to be enlarged in size.

Right Side – 3 vessel umbilical cord seen. The umbilical vein shows pulsatile flow. The umbilical artery shows normal waveform. No reversal seen. Two separate umbilical cord insertions seen. **There is a small vessel communicating between the two umbilical cords.**

Colour Doppler study(Scanned photocopy)



Impression: Monochorionic twin pregnancy with one live fetus and another deformed fetus with no cardiac activity Most likely secondary to twin-twin transfusion sequence. Normal color doppler study of the live fetus.

Delivery Notes: Both babies delivered spontaneous vaginally. 1st Male Baby delivered weight 1.70kg alive, by cephalic presentation. 2nd Male Baby delivered by cephalic presentation at weight 2.25kg with Omphalocele and hydrops, with short hands and legs (Photograph). Placenta delivered spontaneously. It was single with two cords Placenta monochorionic and diamniotic. Patient refused autopsy of malformed fetus

Patient developed haematuria on 2nd post natal day. Her Platelet count was 35,000/min. She was given two units of fresh blood transfusion and two platelets transfusion, haemostatic treatment. Other coagulating profiles were within normal limits. Haematuria regressed after 6th postnatal day. Patient with her baby discharged in good condition on 13th post natal day.

Figure: - Showing Monster baby

Discussion: This rare case with malformed and dead fetus is at risk of subsequent death in the surviving twin and it is six-fold greater in same sex twins. Early demise does not increase risk of death in surviving fetus in first trimester..Later in gestation the death of one fetus could trigger coagulation defects in mother as in above case. If non living tissue remains in uterus the mother is at risk for coagulation problems caused by proteins from dead tissues released into the mother's blood stream. This can cause a disseminated intravascular coagulopathy that can kill the mother and always result in the death of remaining baby. But this happens rarely, because after the death of one twin fetus there is progressive fall in fibrinogen concentration and rise in fibrinogen degradation products. These block the escape of thromboplastin from fetus to placenta and into maternal circulation and thus prevent D.I.C. So the surviving fetus when delivered near term is healthy, has normal plasma fibrinogen, fibrin degradation products level and platelet count at birth. As such cases occur in twins. The real ability lies in early diagnosis, when the onset of this condition occurs before 26 weeks of gestation there is associated fetal loss, perinatal death, and subsequent handicap in survivors[6-8]. If undiagnosed and untreated early T.T.S. perinatal mortality

exceeds 90% [9], and more than 30% of survivors have associated neurodevelopment anomalies[10]. Diagnosing a hydrops and with intra-uterine death can save the mother from life threatening complication of disseminated intravascular coagulation. Fortunately, this case was saved in spite of diagnosis at twelfth hour. Twin to twin transfusion syndrome (TTTS) cannot be prevented but an early diagnosis of this disorder in an identical twin pregnancy can possibly save one or both babies. This can be detected in the early stage of pregnancy by ultrasound scanning and Doppler velocimetry[11]. A careful ultrasonographic monitoring of both twins every fortnightly in last trimester may be a key to success

Conclusion: In twin to twin transfusion syndrome treatment is laser ablation of anastomosis and the survivor has neuro developmental delay which requires long term follow up.

References:

1. Friedrich Schatz, in 1875. [1.] ^ Jump up to:[a] [b] Johnson, A. & Moise, K., J. "Improving Survival in Twin-Twin Transfusion Syndrome. Contemporary OB/GYN December 2006"(PDF).
2. Benirschke K. The placenta in twin gestation. Clin obstet Gyecol 1990; 33:18-31.
3. Czuba B. Wtach A, Borowski D, Sodowski K, et al; Actual treatment options of twin to twin transfusion syndrome Ginekol Pol. 2006 Apr;77(4):317-22. (Article in Polish).
4. Anita Kant, Neural Tube defects of Monozygotic Twins, Journal of Obstetrics and Gynaec India: June 2000; 50(3): 90.
5. BrunerJP, Rosemond RL: Twin-to-twin transfusion syndrome: subset of the twin oligohydramnios-polyhydramnios sequence.Am J.Obset Gynecol 169:925, 1993.
6. Weir PE Rttern GJ Beischer NA Acute polyhydrammnios os a complication of monozygous twin pregnancy .Br J obstet Gynaecol 1979, 86:849-53.
7. Laroche JC Droulle P,.Delezoide AL , Narcy F,Nessman C.Brain damage in monozygous twins.Biol Neonate 1990;57:261-78.
8. Gonsoulin W, Moise KJ Jr., Krishon B, Cotton DB, Wheeler JM, Carpenter RJJr. Outcome of twin – twin transfusion diagnosed before 28 weeks of gestation. Obstet Gynecol 1990;75:214-6
9. Haverkamp F, Lex C, Hanisch C, Fahnenstich H, Zerres K. Neurodevelopmental risks in twin to twin transfusion syndrome : preliminary findings .Eur J Paediatr Neurol 2001;5:21-7
10. Ville Y,HecherK,Gagnon A , Sebire N , Hyett J, Nicolaides K Endoscopic laser coagulation in the management of severe twin to twin transfusion syndrome. Br J Obstet Gynaecol 1998;105:446-53
11. Dashe JS, Fernandez CO, Twickler DM .Utility of Doppler velocimetry in predicting outcome in TRAP sequence. Am j Obstet gynecol 2001; 185:135-9

Conflict of interest: None
Funding: None