Case Report:

Twin to twin transfusion syndrome

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Abstract:
Twin-to-twin syndrome is commonly seen in monochorionic twins due to arteriovenous anastomoses which occurs in 15% cases. The recipient twin is large plethoric while donor twin is small, anemic and malnourished but both are equally at risk. The donor twin is usually anemic so requires packed cell transfusion while recipient twin is usually polycythemic and may need partial transfusion. Herewith we reported a case of twin to twin transfusion syndrome in inborn delivery at our rural setup. The babies were treated as per protocol.

Key-words: Twin to twin transfusion syndrome, anemic twin

Background:
Twin-to-twin transfusion syndrome (TTTS) is the result of an intrauterine blood transfusion from one twin (donor) to another twin (recipient). TTTS only occurs in monozygotic (identical) twins with a monochorionic placenta[1]. The donor twin is often smaller with a birth weight 20% less than the recipient's birth weight. The donor twin is often anemic and the recipient twin is often plethoric with hemoglobin differences greater than 5 g/dL[2]. It is estimated to occur in 5-10% of identical twin pregnancies. Because the recipient twin has more blood, he/she also urinates more and has more amniotic fluid. The donor twin has less amniotic fluid; sometimes there is so little fluid that the fetus appears on ultrasound to be stuck in place on the wall of the uterus (known as "stuck twin phenomenon").[6] Pregnant mother - a rapidly enlarging abdomen over 2-3 weeks, as the amniotic fluid of the recipient twin builds up; premature labor, and premature rupture of membranes (water breaks early)[3]Donor twin - Small for gestational age, 10-20% smaller than recipient twin, pallor (due to anemia), poor circulation

Recipient twin - Large for gestation age, 10-20% larger than donor twin, ruddy (red) skin and jaundice (due to extra blood). Twin-to-twin transfusion syndrome can range from mild to severe. It can occur at any point during the pregnancy, even at birth (once one umbilical cord has been clamped after delivery, the other twin may get a rush of extra blood). The donor twin is usually anemic so requires packed cell transfusion while recipient twin is usually polycythemic and may need partial transfusion.[5]
We reported a case of twin to twin transfusion syndrome in inborn delivery at our rural setup. The babies were treated as per protocol.

**Case report:**
A 27 year old lady G2P1L1 delivered twins by vertex vaginal delivery at 37 weeks 2 days of gestation. 1st twin was a male child of 1903gm weight and pale. While the 2nd twin was also male child of 1930 g weight and plethoric. 1st twin: Hb- 10.9 g/dl, PCV-38 while 2nd twin- Hb- 21 g/dl, PCV- 58. On day 2 of life, the polycythaemic recipient needed partial exchange due to clinically symptomatic features of polycythemia, (increased plethora, peripheral cyanosis and hypoglycaemia (38mg/dl) substantiated by Hb= 24gm/dl and PCV 66% The other twin was given a packed cell transfusion with Hb rising to 13.5 gm/dl. Both babies were given appropriate supportive management and discharged on day 12 of life. Follow up of the neonates showed normal growth & development.

**Treatment:**
The polycythemıc twin was given partial exchange transfusion on day 2 of life. While the anemic twin was given PCV transfusion at 15 ml/kg. The septic screen showed CRP positive so babies were started on IV Antibiotics (ampicillin, cefotaxim). The plethoric twin needed SSPT for 4 days. After completion of 7 days of antibiotics the babies were clinically stable so discharged on day 12 of life.

**Follow-up :**
Both babies were evaluated on Day 19 of life. They were clinically stable and thriving well. Immunisation was ascertained and multivitamins started.

**Discussion:**
Twin-to-twin transfusion syndrome (TTTS) is a complication of monochorionic twin gestations and is one of the most lethal conditions in perinatal medicine. TTTS is thought to result from an unbalanced net transfusion of blood between one twin, the donor, and the other twin, the recipient, via placental vascular anastomoses. In the 19th century, Friedrich Schatz, a German obstetrician, extensively studied the twinning process and was the first to speculate on the relationship between vascular anastomoses and the development of TTTS. In TTTS with double survival, recipient twins are at risk for life-threatening cardiovascular complications such as hypertrophic cardiomyopathy and right ventricular outflow tract obstruction, whereas donor twins are at risk for acute or chronic renal failure due to chronic renal hypoperfusion.[9] Twin gestations represent 1 to
2% of all pregnancies. Two thirds of twin gestations are dizygotic (therefore always dichorionic). One third of twin gestations are monozygotic and may give rise to either monochorionic or dichorionic placentation. Only monochorionic twins are at risk for developing TTTS. About 10 to 15% of all monochorionic twin pregnancies develop TTTS.

The presence of placental vascular anastomoses is a conditio sine qua non for the development of TTTS. Three types of anastomoses have been documented: from artery to artery, from vein to vein and from artery to vein.[11] Arterio-venous anastomoses are unidirectional and are referred to as “deep” anastomoses since they proceed through a shared placental cotyledon, Whereas arterio-arterial and veno-venous anastomoses are bi-directional and are referred to as “superficial” since they lie on the chorionic plate.[8]

Morbidity in TTTS includes neurological, cardiovascular and renal complications.

1. **NEUROLOGICAL**: Hypoxic-ischemic damage caused by cerebral hypoperfusion is cause in donor twins, whereas hyperviscosity and polycythemia causing vascular sludging is cause in the recipient. Donors and recipients are equally at risk for adverse neurological outcome. Cerebral lesions include periventricular leucomalacia, cerebral white-matter cysts, severe intraventricular hemorrhage, cerebral infarct, ventricular dilatation, cerebral atrophy.[10]

2. **CARDIOVASCULAR**: They occur more frequently in monochorionic twins than in singletons population and is found mainly in recipient twin. It includes fetal hypertension, hypertrophic cardiomyopathy, tricuspid regurgitation, left chamber myocardial infarction, pulmonary artery calcification and right ventricular outflow tract obstruction.[7]

3. **RENAL**: These are seen in donor twins in and include renal cortical necrosis and fibrosis, transient renal insufficiency and hematuria, acute renal failure requiring long-term peritoneal dialysis, or permanent tubular dysfunction with polyuria due to renal tubular dysgenesis.[9]

Baby’s parents have given the written informed consent for the publication of images and other information concerned to this case report. I am grateful to the parents for giving permission to publish this image for academic purpose.

TTTS usually leads to considerable weight difference at birth between the twins. But in this case, there was hardly any difference in the birth weights despite all the features of TTTS. This was probably due to twin to twin transfusion late in the gestational period.

Message from here …..

- Early detection and high index of suspicion is necessary for the appropriate management of TWIN-TWIN TRANSFUSION.
- The treatment of TTTS if delayed can lead to lethal complications in both donor as well as the recipient twin.
REFERENCES:


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