Tetralogy of Fallot complicated by Pregnancy Induced Hypertension

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Tetralogy of Fallot (TOF) is a congenital cyanotic heart disease which, if remains uncorrected by surgery, associated with significant morbidity and mortality. Many patients survive beyond childhood without surgery due to physiological adaptations in the form of shunts. Pregnancy in such patients adds to this physiological demand of the heart.

Women with uncorrected TOF are one of the under-reported cases needing good clinical acumen for its timely diagnosis and management as these patients suddenly take a downhill turn during pregnancy. We hereby chose to describe such an uncommon case in our ward, wherein the mother afflicted with TOF was complicated by PIH and subsequently underwent spontaneous premature vaginal delivery with low—birth weight neonate.

Tetralogy of Fallot (TOF) is a congenital heart disease(CHD) accounting 5 – 6% of congenital malformations, characterized by intra-cardiac right to left shunt due to ventricular septal defect (VSD), overriding of aorta, pulmonary outflow obstruction and right ventricular hypertrophy.1

When left uncorrected 25-35% die in the first year of life, 40-50% die by the age of 4, 70% by 10 years and 95% by 40 years. But after corrective surgery >85% survive.2

A thirty year old G2P1 lady known to have Tetralogy of Fallot, not surgically corrected, was referred to our hospital at 34+2 weeks period of gestation for persistently elevated BP (blood pressure) with swelling of bilateral lower limbs for 3 weeks. She had quit her diuretic medications about 11 years back without medical advice.

Her first pregnancy was uneventful. She had a normal vaginal delivery at term at home. The child was healthy and free from congenital diseases, especially cardiac disease. In her second pregnancy, she became cyanotic with SpO2 of 85% with oxygen via nasal cannula. On further examination she had elevated BP, clubbing and bipedal edema and a systolic murmur in the parasternal area. On pelvic examination the patient was not in labor at the time of admission. She was put on prophylactic antibiotics and anti—hypertensive drugs. Hemoglobin was 9.5 gm% and her urine albumin was 2+ while other routine laboratory parameters were within normal limits.

Electrocardiography indicated right ventricular hypertrophy with no evidence of arrhythmia. Echocardiography revealed a large VSD of ~2cm, 35% overriding of aorta, severe critical PS, RVH and LVEF ~57%. An obstetrical ultrasonographic scan showed no fetal cardiac anomalies but suggested IUGR. Modified bio-physical profile was normal.

Within four hours of admission, patient went into spontaneous preterm labor and had a vaginal delivery without instrumentation. Baby was a preterm male of about 1.3 kilograms with poor Apgar score. After delivery, curettage was done for retained placental bits.

Postpartum period of the mother was uneventful. The neonate, however, developed neonatal sepsis at few hours of birth with many episodes of neonatal seizures and was under intensive care with mechanical ventilation but died after four days.

Poor prognostic factors in TOF include maternal hematocrit greater than 60%, oxygen saturation less

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than 80%, and clinical history of syncopal attacks which is believed to give rise to in-utero growth restriction of 36% of pregnancies if there is cyanotic heart diseases. Cardiac complications like heart failure, thromboembolic events, coagulopathy, polycythemia, arrhythmias, endocarditis and chronic hypoxia compound the risk obstetrical complications like pre-eclampsia, eclampsia, preterm labor and postpartum hemorrhage.

**Conclusion:**
This uncorrected case of TOF highlights the need for vigilant monitoring of mother throughout pregnancy. Compounding stress factors on the heart may increase risk of maternal morbidity to mortality. Any complication that arises can compromise the materno-placental circulation resulting in different adverse fetal outcomes. In our case it was low birth weight baby due to pregnancy induced hypertension and eventually neonatal death.

**References**

58