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"Anaesthetic Challenges in A Case of Uncorrected 'Tetralogy of Fallot' With Severe Preeclampsia Posted For Emergency Caesarean Section"

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Abstract: *'Tetralogy of Fallot' is the most common cyanotic heart disease and without surgical correction around 50% of children with TOF die within the first few years of life and survival beyond 30 years without surgery are uncommon. Uncorrected cyanotic heart disease in pregnancy carries a significant risk both for mother and foetus. With several anatomical defects and its physiological impacts; anaesthesia in such a patient become challenging. We reported the anaesthetic management of a patient with uncorrected TOF with severe preeclampsia posted for emergency caesarean section.*

Keywords: *Tetralogy of Fallot, pre-eclampsia, pregnancy, caesarean section*

I. Introduction

Tetralogy of fallot is the most common form of cyanotic heart disease and accounts for 3.5% of all cases of congenital heart disease (1:3600 live births in the UK). The cause is mostly unknown but some chromosomal abnormality may be associated [(1)] .Without surgical intervention around 50% of children with TOF die within the first few years of life and survival beyond 30 years is uncommon [(2)]. Women with uncorrected TOF do poorly during pregnancy and maternal mortality approaches 10% [(3)]. Stillbirth of 14% and foetal growth retardation of 36% of pregnancies in women with cyanotic heart disease has been reported [(4)]. We reported a case of uncorrected TOF with severe preeclampsia posted for emergency caesarean section.

II. Case report

A 23 year female of low socio-economic status married for seven years, 3rd gravida with 34 weeks of pregnancy was admitted with complaint of pain abdomen and pedal swelling for last one week with history of two spontaneous abortions in the past at 10 weeks & 14 weeks of pregnancies. The patient had exertional breathlessness at childhood & adolescent age but never consulted any physician till loss of two pregnancies after marriage. She was diagnosed as a case of 'Tetralogy of Fallot' and was advised for surgical correction but they denied the same due to monetary problem. She was on oral Propranolol 40mg once daily for last 3 years. In current pregnancy she developed severe pre eclampsia for which she was given oral labetalol 100mg thrice daily for last 3 days. She was given prophylactic dose of inj. Magnesium sulphate.

On examination she was found to be dyspnoeic. She had clubbing & bilateral paedal oedema. Her pulse rate was 104/min, BP 158/102 mm Hg and respiratory rate 22/min. On auscultation a clear chest with loud S_2 and harsh pan systolic murmur was heard most loud over the pulmonary area. A thrill was felt along the left sternal area.

Her investigation showed Hb 13.5gm%, PCV of 40.3, BT of 1 min, CT of 7 min. Blood sugar- 83 mg/dl (fasting), Serum creatinine of 0.9mg/dl. ECG showed RVH & right axis deviation. Echocardiography revealed large VSD with approx. 50% overriding of aorta, RVOT gradient of 80 mmHg with good biventricular systolic function. (Figure 1) Pre pregnancy chest X-ray showed boot shaped heart. (Figure 2)

Emergency caesarean section was decided because of foetal distress. General anaesthesia was planned. Opinion from cardiologist & high risk consent was taken. Inj. Ranitidine 50mg & inj. Metoclopramide 10mg was given IV 30min prior to operation. Preloading was done with 500ml of Ringer's lactate. Inj. Glycopyrrolate 0.2mg given IV. Co-induction was done with inj. Ketamine 50mg IV and Inj. Thiopentone Sodium 2.5% 150mg IV. Endotracheal intubation was done with rapid sequence after inj. Succinyl Choline 75mg IV. 100% oxygen was administered till delivery of the baby then 50% N2O was mixed with O2. The patient was monitored for ECG, NIBP, pulse rate, SpO2. The baseline SpO2 was 88% in room air. The weight of the baby was only 850 gm i.e. severe LBW and APGAR score at 1st & 5th min was 7 & 8 respectively. Inj. Oxytocin 10 units were given in IV infusion very slowly. IM oxytocin was avoided. The patient was maintained with inj. Fentanyl 50mcg IV, inj Atracurium 25mg and 0.2-0.4 %(v/v) Isoflurane. A total of 600ml RL was given intra-operatively. SpO2 was maintained around 92-94% throughout the operation. At the end of the surgery the patient was reversed with Inj. Neostigmine 2.5mg IV & Inj. Glycopyrrolate 0.5mg IV and the trachea was

extubated. Infusion Paracetamol 1gm IV was given slowly for immediate postoperative pain control. BP throughout the procedure was maintained between 158/106 and 148/96mmHg, and pulse rate ranged from 98-110/min. There was no cyanotic spell, no significant fall or rise in BP intraoperatively. Inj. Phenylephrine was kept ready to prevent hypotension. The patient was closely monitored in the immediate postoperative period in the HDU. She was given the 2nd & 3rd dose of inj. MgSO4. Oral Labetalol was continued till 3rd day postoperatively. The postoperative period was more or less uneventful and she was discharged on the 8th postoperative day.

Figure-1(Echocardiography)

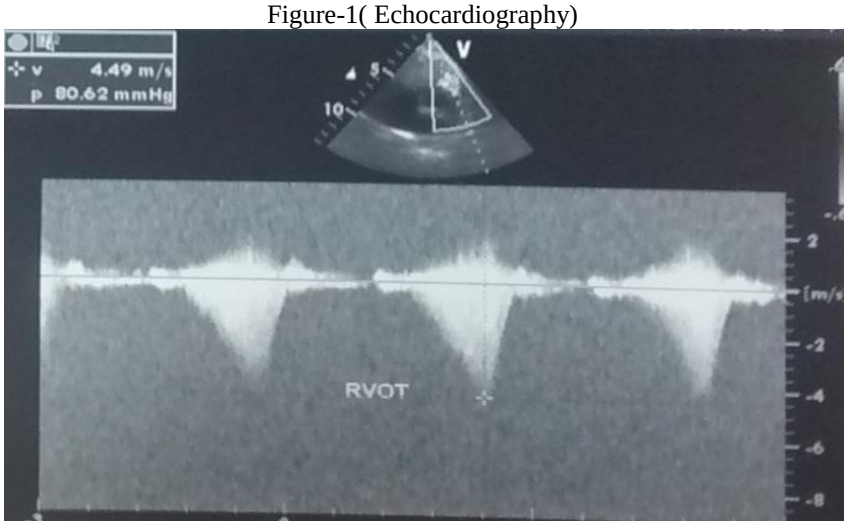
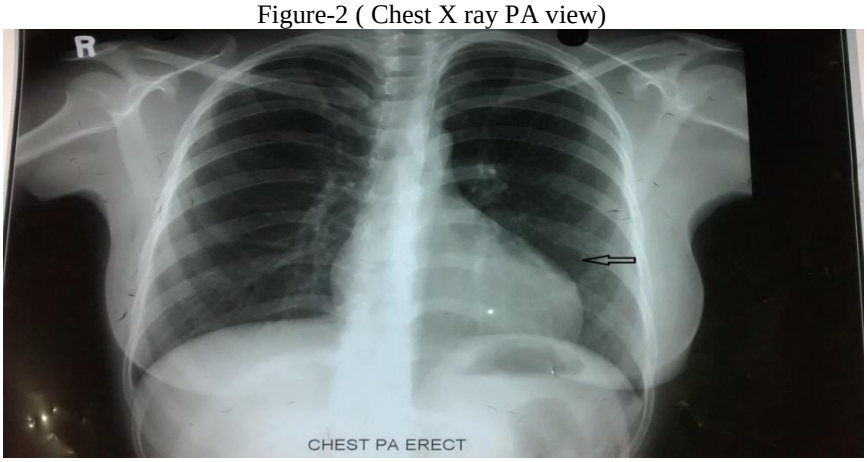


Figure-2 (Chest X ray PA view)



III. Discussion

The four components of TOF are malaligned ventricular septal defect (VSD), an abnormally positioned aortic valve above (override) the ventricular septum, right ventricular outflow tract (RVOT) obstruction, right ventricular hypertrophy (RVH) [(1)]. The relationship between resistance of blood flow from ventricles into aorta and into the pulmonary vessels plays a major role in determining the hemodynamic and clinical picture. When the obstruction to pulmonary vessels is severe, the pulmonary blood flow is reduced markedly and a large volume of desaturated systemic venous blood is shunted from right to left across VSD [(5)].

In pregnant patients with TOF, the decrease in peripheral resistance that accompanies pregnancy augments right to left shunt and may exaggerate maternal cyanosis, which poses risks for both mother and foetus [(6)]. Any disease complicated by severe maternal hypoxaemia is likely to lead to miscarriage, preterm delivery, or foetal death. When hypoxaemia is intense enough to stimulate a rise in haematocrit above 65%, pregnancy wastage is 100% [(7)].Adverse maternal events may be associated with left ventricular dysfunction, severe pulmonary hypertension, and severe pulmonary regurgitation with RV dysfunction.

If TOF remains uncorrected, pregnancy presents serious risks, including maternal mortality. Pre-existing pulmonary hypertension is a concern. In addition, elevated cardiac output leads to increased venous return to hypertrophic right ventricle. These changes, together with decreased systemic vascular resistance increase the right to left shunt. Oxygenation decreases, haematocrit rises, and cyanosis worsens, further stressing

an already compromised system. Risk factors worsening the prognosis include prepregnancy haematocrit exceeding 65%, history of cardiac failure or syncope, cardiomegaly, RV pressure exceeding 120 mm Hg or strain pattern of ECG or saturation less than 80% [(8)]. The magnitude of right to left intracardiac shunt can be increased by decreased systemic vascular resistance, increased pulmonary vascular resistance or by increased myocardial contractility [(9)].

Our goal during anaesthesia was to maintain systemic vascular resistance and to avoid any decrease in peripheral vascular resistance as any of these changes would result in increased right to left shunt. Both general and regional anaesthesia have been employed successfully in pregnant women with TOF [(10)]. Regarding caesarean section general anaesthesia (GA) is probably the technique of choice [(11)]. Hence, we planned for GA. We co-induced our patient with inj. Ketamine as a dose of 1mg/kg with inj. Thiopentone as a dose of 3mg/kg so that BP doesn't raise much but maintain the SVR. Though invasive arterial BP monitoring is the standard method of choice for BP monitoring, but we couldn't do that due non availability. We ventilated the patient's lungs avoiding excessive positive airway pressure, as intermittent positive pressure ventilation can decrease pulmonary blood flow by increasing pulmonary vascular resistance. 100% O2 was given till the birth of baby, and after delivery N2O 50% in O2 was given, keeping a close watch on oxygen saturation. N2O increases pulmonary vascular resistance, but this potentially adverse effect is more than its beneficial effects on systemic vascular resistance (no change or modest increase) [(9)]. So we limited the inspired concentration of N2O to 50%.We preloaded our patient with 500 ml of lactated ringer's solution so as to maintain intravascular volume during surgery as acute hypovolemia tends to increase right to left intracardiac shunt. We avoided IM Oxytocin rather we gave it very slowly with infusion as IM bolus might reduce peripheral vascular resistance. There was no adverse intraoperative event or any significant alteration in blood pressure or decrease in oxygen saturation below 90%.

IV. Conclusion

Anaesthetic management of patients with TOF requires thorough understanding of anatomical defects and its physiological impacts, effects of different anaesthetic agents on body system. It necessitates an integrated approach by a team consisting of obstetrician, cardiologist and anaesthesiologist among others.

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