



BMH Med. J. 2022;9(1):12-14. **Case Report**

A Case of Uncorrected Tetralogy of Fallot for Caeserian Section: An Anaesthetic Challenge!

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Introduction

Uncorrected cyanotic heart disease carries a high risk in pregnancy for both mother and fetus. TOF accounts for 5-6% of cases of congenital heart disease [1]. A review of 57 pregnancies with uncorrected Tetralogy of Fallot (TOF) showed a fetal mortality of 22% and a maternal mortality of 7% [2]. We report a case of parturient with uncorrected TOF managed successfully under Combined Spinal Epidural (CSE) anaesthesia for lower segment caeserian section (LSCS).

Case report

A young second gravida, known case of congenital cyanotic heart disease/TOF and pulmonary atresia came to our hospital at 16 weeks gestation. Her first pregnancy was IUD at 8 months gestation. She remained largely asymptomatic and was on medical follow up. She had dyspnoea on exertion class II, not associated with chest pain or giddiness. On examination she had central cyanosis grade 2 clubbing, heart rate (HR) of 116/min, blood pressure (BP) 140/90 mm Hg and oxygen saturation (SPO₂) of 84% on room air. Cardiovascular system examination revealed a pan systolic murmur grade 3/6 in pulmonary and aortic areas. There were no features of heart failure. Electrocardiogram showed sinus tachycardia with poor R wave progression. ABG was normal except for a pO₂ of 64 mm Hg and SO₂ 94%. Echocardiography demonstrated large conoventricular ventricular septal defect with bidirectional shunt, large segment pulmonary atresia, right aortic arch, 50% aortic override, multiple major aorto-pulmonary collateral arteries and good biventricular function. Her hemoglobin and hematocrit was 14.1 gm% and 41.8% respectively.

USG at 29 weeks revealed absent diastolic flow in the fetus and emergency LSCS was planned. High risk consent was obtained. Inj Cefazolin 2g was administered one hour prior to surgery as prophylaxis against infective endocarditis. She was premedicated with intravenous (IV) ranitidine 50 mg and metoclopramide 10 mg. In the operating room, noninvasive blood pressure (NIBP), electrocardiogram and SPO₂ were monitored. Her NIBP and SPO₂ were 170/80 mm Hg and 84% respectively. O₂ was administered by polymask at 6 L/min.

We planned for a combined spinal epidural anaesthesia. Under USG guidance and local anaesthesia right internal jugular catheter and left radial artery were cannulated for invasive monitoring. Based on central

venous pressure (CVP), HR and BP response Ringer's lactate was administered slowly. CVP was maintained at 6-8 mm Hg and BP was 170/80 mm Hg. In lateral position, an epidural catheter was inserted at T12-L1 intervertebral space. Then using a 25 G Quinke spinal needle subarachnoid block was given at L3-4 space with 0.5 ml of 0.5% hyperbaric bupivacaine. She was then positioned supine. A wedge of 10 cm height was placed under right buttock. We could attain a sensory block (to pin prick) to the level of T12 after 2 minutes. Then we administered 3 ml of 0.5% ropivacaine via epidural catheter and 2 ml more after 5 minutes. Level of sensory block was T7 15 minutes later. We intentionally kept sensory block not higher than T6 to avoid any dramatic hemodynamic fluctuations. A male neonate was delivered who cried soon after birth. APGAR was 9 at 1 minute and 10 at 5 minutes. Oxytocin 10 units was given as slow IV infusion. We also gave Inj Tranexemic acid 500 mg. SPO₂ remained between 88-92 intraoperatively. Her hemodynamic parameters remained stable throughout surgery.

We administered a total of 600 ml of IV fluids. Estimated blood loss was 750 ml. Urine output was 125 ml. The patient was transferred to ICU and was closely monitored for HR, BP and SPO₂. Her hemodynamic parameters remained stable in the postoperative period. Postoperative analgesia was provided in the form of continuous epidural infusion of 0.2% ropivacaine with 2 mcg/ml fentanyl at the rate of 4-6 ml/hour. She was discharged from ICU to ward on second post operative day and discharged home on sixth post operative day.

Discussion

As described by the French Physician Etienne-Louis Arthur Fallot in 1888, Tetralogy of Fallot consists of four anatomical components: (1) Ventricular septal defect (VSD), (2) an abnormally positioned aortic valve above (override) the ventricular septum, (3) Right ventricular outflow tract obstruction (RVOT) and (4) Right ventricular hypertrophy. Uncorrected group presents a severe challenge, in issues related to long term effects of chronic hypoxia. They have reduced exercise tolerance and are vulnerable to ventricular arrhythmias or sudden death secondary to chronic pulmonary regurgitation and problems related to abnormalities in RV physiology [3,4]. When there is severe obstruction to pulmonary blood vessels, desaturated systemic venous blood is shunted from right to left across VSD. Severe cyanosis and erythrocytosis occurs. Symptoms of systemic hypoxemia will be present [5].

Factors causing right to left shunt are (1) increased pulmonary vascular resistance (PVR), (2) decreased systemic vascular resistance (SVR) and (3) increased myocardial contractility [6]. Amount of shunting is determined by ratio of SVR to PVR. Reduced peripheral resistance that accompanies pregnancy augments the right to left shunt causing increased risks for both mother and fetus [7]. Maternal hypoxemia can lead to preterm delivery, miscarriage or fetal death. Maternal complications include left ventricular dysfunction, severe pulmonary regurgitation with RV dysfunction. If hypoxemia is severe enough to cause a rise in HCT above 65 %, pregnancy loss is 100% [8].

Choice of anaesthesia: General versus Intravertebral

Generally, anaesthesiologists choose general anaesthesia (GA) for anaesthetic management of patients with congenital heart disease due to rapid sympathetomy and vasodilatory effects in intravertebral anaesthesia which may result in more hemodynamic instability and worsen right to left shunt. However GA is associated with some disadvantages. For instance, induction agents can depress myocardial contractility and decrease peripheral vascular resistance. Also, increase in intrathoracic pressure by intermittent positive pressure ventilation can reduce venous return, thereby decreasing cardiac output [9-11]. In addition, GA can also greatly increase risk of aspiration in pregnant women.

Our goal during anaesthesia was to prevent the increase in right to left shunt. So we planned for a combined spinal epidural technique which provides a better hemodynamic stability. Spinal anaesthesia in conventional dose is associated with a drop in peripheral vascular resistance. A low dose spinal anaesthesia provides adequate surgical anaesthesia, improved recovery time, but no difference in maternal cardiac index when compared to conventional dose spinal anaesthesia [12]. CSE offers a rapid reliable onset coupled with capacity

to augment or prolong the blockade through epidural catheter. It allows titration of level, density and duration of anaesthesia. Further, we avoided oxytocin bolus dose as it will affect peripheral vascular resistance. Adrenergic agonist phenylephrine was kept ready but was not used. There was no fall in blood pressure or saturation or any other adverse intraoperative event.

In our case, combined spinal epidural anaesthesia was administered to a parturient with uncorrected TOF, ensuring good outcome for both mother and baby and meeting the need for emergency surgery.

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