Anaesthetic management for excision of Left temporal abscess in a case of Tetralogy of Fallot with severe perioperative hypoxia: A case report

Nitish Saini¹, Pramila Soni², Durgesh Agrawal¹, Sonali Dhawan³, Aditi Maheshwari⁴

¹- Junior resident, Sardar Patel Medical College, Bikaner
²- Associate Professor, Sardar Patel Medical College, Bikaner
³- Professor and Head of Department, Sardar Patel Medical College, Bikaner
⁴- Junior resident, Sardar Patel Medical College, Bikaner

Corresponding author - Aditi Maheshwari

ABSTRACT: Tetralogy of Fallot is a cyanotic congenital heart disorder which includes ventricular septal defect, right ventricular outflow tract obstruction (pulmonary stenosis), overriding of aorta and right ventricular hypertrophy.[1] The main goal for anaesthetic management in these patients is to maintain or increase the systemic vascular resistance, reducing the pulmonary vascular resistance and provide mild cardiac depression.[2] Uncorrected TOF in patients leads to chronic hypoxia and decreased pulmonary blood flow which results in considerable alteration in the physiology of body. Because of paradoxical embolism and absence of pulmonary phagocytic clearance of pathogens in these patients there is increase chances of brain abscesses. Here, we present a case of excision of left temporal abscess under general anaesthesia, in a 10 year old child of uncorrected TOF.

I. CASE REPORT

A 10 year old male child was referred for pre anesthesiacheckup for excision of brain abscess. He presented with complaints of high-grade fever with altered sensorium. He was on meropenem, vancomycin, metronidazole. He was born at full term and there was no history of delay in milestones. He has presented with history of breathlessness on mild to moderate exertion and history of squatting was present.

On examination child was of 22kg of weights and sitting in squatting position. He had fever, clubbing, central cyanosis with heart rate of 130 per minute, respiratory rate of 18 per minute, spO2 54% on air and blood pressure of 96/60 mm of Hg. Airway was normal with mallampati grading of class II. On cardiovascular examination he had ejection systolic murmur. Respiratory system examination was normal with bilateral air entry present. Patient was oriented to time, place and person.

His haemoglobin was 13.3g/dl with haematocrit of 49.9% with neutrophilia (70.3%) and platelet count of 1.48lakh/mm3. Other routine investigations were normal. Chest X-ray was normal. Electrocardiograph showed right ventricular hypertrophy with right axis deviation. ECHO revealed adult TOF with large VSD (sub aortic) of 20mm in size, aortic override of >50%, non-restrictive right to left shunt, severe pulmonary stenosis, and left aortic arch. Computerised tomography, pulmonary angiography revealed relatively thin pulmonary arteries with dilatation of ascending aorta with overriding of aorta and mild cardiomegaly.

Magnetic resonance imaging brain showed a well defined lesion in left temporal region of size 30X23X29mmand mild meningeal enhancement in left temporal region. Abscess was not resolving with antibiotics so excision of abscess under general anaesthesia was planned. He was certified asASA IV (E) grade risk. A bed in paediatric intensive care unit was reserved for postoperative care. He was hydrated with Ringer lactate solution at 2 ml/kg/hr.

A cardiologist opinion was taken for perioperative management and it was decided to do the surgery under very high-risk consent. The boy was kept nil per oral for 6 hours duration. All intravenous lines were de-aired and inotropes, vasodilators and the defibrillator kept ready. Standard monitoring was started.

Intravenous esmolol 10mg was given slowly to reduce heart rate. Soda bicarbonate...
10mEq was given slowly. Pre-medication consisted of intravenous midazolam 1 mg and fentanyl 40 μg injected slowly. He was preoxygenated and anaesthesia was induced with etomidate 4 mg given slowly and atracurium 10 mg and intubated with uncuffed endotracheal tube of 5.5mm. After confirming Bilateral air entry, tube was fixed at 14cm. Anaesthesia was maintained with 0.8%-1% sevoflurane with 100% oxygen. Esmolol infusion was started at 150 μg/kg/min. Atracurium with maintenance dose of 2.5 mg was given stat to maintain muscle paralysis. Intraoperative NIBP reading varied between 86–110 mmHg and 48–72 mmHg. SPO₂ reading varied between 55% and 94%. This was treated with fluid bolus 10 ml/kg, empirical sodium bicarbonate 1 mEq/kg for hypotension associated with desaturation. Optimal ventilation was given through modified Jackson-Rees circuit. Drainage of abscess with excision of the covering layer was done and the surgery lasted for 2 hr. Intravenous paracetamol 300 mg and ondansetron 2 mg were administered. Ringer’s lactate solution was used for maintenance. The paralysis was reversed after spontaneous return of breathing with neostigmine 1mg. Soda bicarbonate 10mEq was repeated just before extubation. Patient was extubated after adequate neuromuscular recovery and shifted to PICU for further care. Post operative vitals after 6 hours were SpO₂ 85% on nasal prongs at oxygen 4L/min, pulse rate 78/min, BP of 110/74mmHg.

II. DISCUSSION

Tetralogy of Fallot is cyanotic heart disease which accounts for 10% of congenital heart disease. Of all the patients with brain abscess and cyanotic congenital heart diseases, TOF is the most common in association (13-70% of the cases).[3] It includes tetrad of ventricular septal defect, right ventricular outflow tract obstruction (pulmonary stenosis), right ventricular hypertrophy, and overriding of aorta. There is increased risk of the perioperative complications and mortality in patients with uncorrected TOF.[4] They can also present with associated noncardiac anomalies such as neurological deformities, musculoskeletal anomalies, and ophthalmological anomalies. Major anaesthetic goals in these patients are to maintain or increase the systemic vascular resistance while reducing pulmonary vascular resistance and prevention of hypercyanotic episodes intraoperatively.

Right to left shunting leads to poor pulmonary perfusion which causes chronic hypoxemia and cyanosis. The compensation includes polycythaemia, vasodilatation, hyperventilation and chronic respiratory alkalosis. Infective Endocarditis prophylaxis is recommended for uncorrected TOF.[5] Adequate hydration perioperatively is necessary as it reduces blood viscosity, sludging and prevent thromboembolism. Hypovolemia can exacerbate the right ventricular outflow tract obstruction if the patient has infundibular stenosis. Maintaining high systemic vascular resistance as compared to pulmonary vascular resistance minimises right-to-left shunting. Prevention of hypoxia, hypercarbia and acidosis is necessary to reduce the increase in pulmonary vascular resistance.[6] Hyperventilation without PEEP helps to decrease pulmonary vascular resistance. As Ketamine is avoided for induction in neurosurgery, adequate sedation and induction with etomidate was our first choice. High dose opioid and benzodiazepine induction were avoided to prevent any postoperative respiratory depression and delayed recovery. Sevoflurane <1 MAC prevents cerebral vasodilation and excessive myocardial depression, which could potentiate right ventricular failure. Esmolol infusion was started to control the heart rate.

These patients are at increased risk of perioperative hyper cyanotic spells. An intraoperative spell is treated with fluid bolus, deepening the anaesthesia plane, opioid, phentylephrine and oxygenation with 100% oxygen to decrease PVR. Dexmedetomidine can also be used to ameliorate cyanotic spell. [7] Early extubation prevents increase in pulmonary vascular resistance due to prolonged ventilation. Post-operative care includes cardiac monitoring, oxygenation, good analgesia, fluid management and prevention of vomiting and seizures.[8]

REFERENCES


