Anaesthetic management for drainage of frontoparietal abscess in a patient of uncorrected Tetralogy of Fallot

INTRODUCTION

Tetralogy of Fallot (TOF) is the most common cyanotic congenital heart disease, characterised by aortic overriding, right ventricular hypertrophy, pulmonary stenosis (PS) and ventricular septal defect (VSD). When left untreated, survival to adulthood is rare.[1] Brain abscesses can occur in them because of paradoxical embolism and absence of pulmonary phagocytic clearance of pathogens.

There are few case reports of uncorrected TOF surviving to adulthood and then presenting for non-cardiac surgeries.[1-3] We present a case of drainage of a large left fronto-parietal abscess under general anaesthesia, in a patient of uncorrected TOF.

CASE REPORT

A 35-year-old male patient was referred for pre-anaesthesia check-up for excision of brain abscess. The patient presented with disorientation and increased sleepiness and no other neurological deficit or signs of raised intracranial pressure. He was on phenytoin, vancomycin, ceftazidime and metronidazole. He had dyspnoea, which gradually worsened from New York Heart Association grade I in childhood to grade IV. There was no history of cyanotic spells. His weight was 52 kg, pulse 74 beats/min, regular, blood pressure 118/72 mm of Hg, SpO_2 of 98% on room air. He was febrile, had clubbing grade III and mild central cyanosis. Airway was normal. He had a normal S1 with grade III ejection systolic murmur in the pulmonary area. Respiratory system examination was unremarkable. His Glasgow coma scale (GCS) was 13/15. Neurological examination was normal except for altered sensorium.

His haemoglobin was 16.7 g/dl with a haematocrit of 49.5%, neutrophilia (75%) and platelet count 2.8 lakh/mm^3. Other routine investigations were normal. Pre-operative arterial blood gas (ABG) showed compensated respiratory alkalosis. Chest X-ray was normal. Electrocardiograph showed right bundle branch block with right ventricular strain. Echocardiography revealed TOF with severe valvular and infundibular PS with a large mal-aligned bidirectional VSD. His PS gradient was 90 mm of Hg. Computerised tomography, chest angiography revealed a right sided aortic arch with overriding aorta, VSD 1 cm diameter, right ventricular wall hypertrophy and infundibular stenosis (0.5 cm). There were no major aortopulmonary collaterals.

Magnetic resonance imaging brain showed a large (3.6 cm × 2.8 cm × 3.7 cm) abscess in left fronto-parietal region, which was not resolving in spite of high dose antibiotics. Drainage of the abscess under general anaesthesia was planned. The patient appeared to be a 'pink Tet'. He was accorded American Society of Anesthesiologists IV (E) grade risk. A bed in cardiac intensive care unit was reserved for postoperative care. He was hydrated with Hartmann's solution at 2 ml/kg/hr.

All intravenous lines were de-aired and inotropes, vasodilators and the defibrillator kept ready. Phenylephrine was loaded. Standard monitoring was started. A 14 gauge and 16 gauge peripherally inserted central line cannulation and left radial artery cannulation were performed for invasive monitoring before induction. The central venous pressure was 4–6 cm of water. Pre-medication consisted of intravenous midazolam 1 mg and fentanyl 100 μg injected slowly. Anaesthesia was induced with thiopentone 200 mg given slowly, titrated to haemodynamics, and rocuronium 50 mg was used for intubation. Dexmedetomidine 0.2 μg/kg/hr infusion was started without the loading dose. Anaesthesia was maintained on sevoflurane (minimum alveolar concentration [MAC] 0.8–0.9) in oxygen: nitrous oxide (N_2O) (50:50) and atracurium infusion (4 μg/kg/min). Two episodes of hypotension were treated with bolus doses of phenylephrine 50 μg. Optimal ventilation without positive end expiratory pressure (PEEP) was provided. Intraoperative saturation ranged from 94% to 100%. Drainage of abscess with excision of the covering layer was carried out; the surgery lasted for 1.5 hr. Intravenous paracetamol 1 g and ondansetron 4 mg were administered. The paralysis was reversed after spontaneous return of breathing and trachea extubated on table. His post-extubation ABG was normal. There was an immediate improvement in GCS with the patient correctly answering all questions. He was shifted to Intensive Care Unit. After an uneventful post-operative course, he was discharged on day 3 and asked to follow-up in the cardiology department.

DISCUSSION

Of all the patients with brain abscess and cyanotic congenital heart diseases, TOF is the most common in association (13–70% of the cases).[4] Prolonged survival with uncorrected TOF is associated with a well-developed left ventricle, mild to moderate PS with adaptations such as systemic-pulmonary collaterals, persistent patent ductus arteriosus or systemic hypertension.[2] The longevity of our patient is explained by a large bidirectional VSD.

Right to left shunting leads to poor pulmonary perfusion resulting in chronic hypoxemia and cyanosis. The compensation includes polycythaemia, vasodilatation, hyperventilation and chronic respiratory alkalosis.[3] Anaesthetic concerns are perioperative haemodynamic instability, cyanotic spells, polycythaemia induced coagulation defects, paradoxical air embolism, fluid and acid base imbalances, congestive cardiac failure, infective endocarditis (IE) prophylaxis and maintaining intracranial dynamics. IE prophylaxis is recommended for uncorrected TOF.[5] Adequate hydration reduces increased blood viscosity, sludging and thromboembolism.[6] Hypovolemia can exacerbate the right ventricular outflow tract obstruction if the patient has infundibular stenosis. Maintaining higher systemic vascular resistance (SVR) relative to pulmonary vascular resistance (PVR) minimises right-to-left shunting. Hypoxia, hypercarbia and acidosis can cause significant increases in PVR. Hyperventilation without PEEP helps to decrease PVR. Ketamine for induction is avoided for neurosurgery. Adequate sedation and slow titration of thiopentone were our choice due to non-availability of etomidate. High dose opioid and benzodiazepine induction were avoided due to short duration of surgery and to facilitate post-operative neurological assessment. N_2O can increase PVR but this is offset by a modest increase in SVR. Minimum 50% FiO_2 should be maintained and so N_2O should be restricted to 50% only. Sevoflurane <1 MAC prevents cerebral vasodilation and excessive myocardial depression, which could potentiate right ventricular failure. Dexmedetomidine, a sedative with minimal respiratory depression, blunts the sympathetic response to intubation and reduces opioid and volatile agent requirement. We started an infusion at the lowest dose after omitting the loading dose to avoid the initial biphasic changes in haemodynamics.[7] It reduces the myocardial oxygen consumption by decreasing the heart rate. Furthermore, use of large dose dexmedetomidine can reduce the SVR resulting in a reversal of shunt flow. Addition of low dose dexmedetomidine to fentanyl reduces the stress response in paediatric patients undergoing cardiac surgeries.[8] Combination of dexmedetomidine and ketamine has been used in two patients of uncorrected TOF for surgeries under general anaesthesia.[6]

These patients are vulnerable to hypercyanotic spells perioperatively. An intraoperative spell is treated with fluid bolus, deepening the anaesthesia plane, fentanyl, phenylephrine and hyperventilation with 100% oxygen to decrease PVR. Dexmedetomidine can also ameliorate cyanotic spell.[9]

Early extubation prevents increased PVR due to prolonged ventilation. Post-operative care includes cardiac monitoring, oxygenation, good analgesia, fluid management and prevention of vomiting and seizures.

CONCLUSION

Successful anaesthesia management for neurosurgery in patients with TOF depends on understanding the pathophysiology of TOF and maintaining the intracranial dynamics. Dexmedetomidine can be a good adjuvant in such cases.

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