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CASE REPORT

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

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ABSTRACT

The birth prevalence of congenital heart disease in India is about 9/1000 with Tetralogy of Fallot (TOF) making up 7-32 % of these cases. TOF is the most common cyanotic congenital heart disease. Children with uncorrected TOF presenting for non-cardiac surgery pose several challenges to the anaesthesiologist due to the altered hemodynamics and mandate a meticulous anaesthetic plan. We report the anaesthetic management of a rare case of a 12-year-old child with uncorrected TOF, who presented to our superspeciality hospital with a brain abscess and underwent a decompressive craniotomy with successful drainage of the brain abscess.

Considering a birth prevalence of congenital heart disease as 9/1000, the estimated number of children born with congenital heart disease in India is more than 200,000 per year with TOF comprising 7-32 % of these cases. About one-fifth of these children are likely to have serious defects, requiring an intervention in the first year of life.[1]

The classic tetrad of TOF is:

1. Pulmonary outflow tract obstruction (stenosis or atresia)
2. Ventricular septal defect (VSD)
3. Overriding aorta
4. Right ventricular (RV) hypertrophy

TOF is primarily caused due to underdevelopment of the sub-pulmonary infundibulum during embryogenesis.

Brain Abscess is a relatively unusual but potentially life-threatening infection of brain parenchyma, which can occur in around 5%–18.7% of the population with cyanotic congenital heart disease.[2] Right to left shunting and hyper viscosity of blood predisposes these patients to brain abscess. Perioperative management of these patients with uncorrected TOF for non-cardiac surgery is challenging for the anesthesiologists owing to the long-term effects of hypoxia and decreased pulmonary blood flow which result in considerable modification of the physiology along with neurological complications.[3]

Anaesthetic goals in such patients is to maintain or increase the systemic vascular resistance, minimize pulmonary vascular resistance and provide mild cardiac depression.[4]

Here we report a case of 12 year old boy with uncorrected Tetralogy of Fallot posted for a non-cardiac surgery.

CASE REPORT

A 12 year old boy presented to the Cardiology outpatient Department of our hospital with history of fever for 15 days and dyspnoea on exertion (Grade 1 NYHA). His metabolic equivalents of oxygen consumption were < 4. There was no recent history of loss of consciousness, seizures or vomiting. He was a known case of Tetralogy of Fallot since birth and was born at full term by caesarean section. There was history of delayed milestones and easy fatiguability. He was immunized for age. He gave history of generalised seizures 5 years back for which he was started on tablet phenytoin 50mg BD but he discontinued that after a while.

He had history of recurrent cyanotic spellsonsquatting and was admitted to hospital for the same 2 years back, started on tablet propranolol 10mg QID which he however discontinued this also after sometime.

On examination, patient was conscious, oriented and co-operative; poorly built (135 cm height) and nourished (28kg weight), grade 2 Clubbing and peripheral and central cyanosis was present.

He was afebrile with pulse rate of 109 bpm regular, blood pressure of 101/62 mmHg in the right arm, with pulse oximetry showed 85-88% oxygen saturation at room air, and respiratory rate of 18 cycles/min.. No abnormality was detected on systemic examination except grade 3 pansystolic murmur heard best in the pulmonary and tricuspid areas. No focal neurological deficit was found on neurological examination.

Haematological workup of the patient including complete blood count, renal function profile, liver function profile and coagulation parameters were normal except RBC count = 7.68 10^6/µl, RDW-CV = 18%, RDW-SD = 62 fl, Haemoglobin = 22 gm/dl, haematocrit = 69.3%, platelet count = 117 10^3/µl, WBC count = 12.31 10^3/µl (Neutrophils : 73.9%) and S. ALP = 341.3 U/L.CECT of the head revealed two early capsulated parietal cerebral abscesses of roughly 3 cm^3 size with contralateral midline shift of 0.9 cm and effacement of left lateral ventricle and subfalcine herniation.

Further evaluation of patient by 2-D Echocardiography revealed features suggestive of TOF physiology - perimembranous ventricular septal defect with right ventricular hypertrophy with 30-40% overriding of aorta with infundibular pulmonary stenosis (gradient 70-75 mmHg) and 40% left ventricular ejection fraction.

He was referred to the Neurosurgery Department for further evaluation for its evaluation.

The patient was planned urgently for decompressive craniotomy under general endotracheal anaesthesia with ASA class IV risk status. NPO orders were followed. 20gauge peripheral intravenous access was secured. Before surgery, infective endocarditis prophylaxis, Inj. Phenytoin 50mg i.v. and Inj. Mannitol 750mg i.v.were given.After securing all ASTM (fullform) standard monitors, patient was premedicated with Inj. Midazolam 0.5mg i.v., Inj. Fentanyl 20mcg i.v. and Inj. Esmolol 5 mg i.v. Anaesthesia was induced with inj. Ketamine 40 mg i.v. and intubated with 5.0 mm ID cuffed portex oral endotracheal tube after Inj. Vecuronium 3 mg i.v. Right femoral central venous catheter was secured and right radial artery invasive blood pressure monitoring initiated. Anaesthesia was maintained with Inj. Dexmedetomidine30mcg i.v. over 20 min. followed by 15 mcg/hr infusion, Sevoflurane MAC 0.8-2.0 with Oxygen: Air ratio 1:2, Inj. Vecuronium 0.3 mg boluses, inj. Ketamine 20 mg i.v intermittently and multimodal analgesia including scalp block, i.v. opioids and acetaminophen. SpO_2 was maintained between 80-90 % and BP titrated with inj. Phenylephrine infusion 300 mcg/hr. Heart rate kept fluctuating between 70-120 bpm. There was a blood loss of about 400 ml and 1000 ml balanced salt solution was infused along with 1 unit packed RBCs. Urine output at the end of the 3 hour procedure was 700 ml.

Patient was reversed from the muscle relaxant at the end of 2 hour surgery and electively ventilated under i.v. sedation. He was successfully extubated next day and discharged after few days.

DISCUSSION

Tetralogy of fallot accounts for about 10% of congenital heart disease and about 50% die during the first year of life. Children who survive this period present with hypoxia, cyanosis, polycythemia, coagulopathies, congestive heart failure and cyanotic spells.[2,5]

Solitary brain abscess constitutes only 0.5% - 6% of the reported cases of brain abscess. The incidence of brain abscess in the population with cyanotic congenital heart disease (CCHD) varies from 5 to 18.5%.

CCHD was also found to be an important predisposing factor for cerebral abscess in 25 – 46% of cases.[6] Tetralogy of Fallot (TOF) is the most common CCHD associated with intracranial suppuration, abscesses being supratentorial, mostly.

Extra cardiac surgeries carry a much higher risk and the an aesthesiologist should be informed about the heart lesion, its altered physiology and an aesthetic implications. Other than the classic tetrad of TOF, patients can also have non-cardiac anomalies including musculoskeletal abnormality, neurological defects and genitourinary defects.[5]

In patients with CCHD, right to left shunting of blood bypassing phagocytic activity of lungs allows direct entry of blood to cerebral circulation. Low perfusion area in brain due to polycythemia leads to tissue hypoxia and acidosis.[7] Microorganisms with shunted blood seeded in such area lead to cerebral abscess. The most important peri-operative concern is the development of cyanotic spells due to spasm of the hypertrophied infundibulum. Factors leading to

infundibular spasm are tachycardia, increased myocardial contractility, decreased systemic vascular resistance (SVR), increased right to left shunt through VSD and systemic blood pressure less than 60 mmHg.[3,6]

The Anaesthetic goals for a case of uncorrected Tetralogy of Fallot posted for a non-cardiac surgery are to avoid hypoxemia, ensure adequate hydration, maintain systemic arterial blood pressure (SVR), minimise additional resistance to pulmonary blood flow (pulmonary vascular resistance) and avoiding sudden increase in systemic oxygen demand (cry, inadequate depth of an aesthesia, seizure, pain, etc).[8]

Induction of Anaesthesia can cause vasodilatation and the resultant fall in SVR can worsen the right to left shunt. We used Ketamine as the induction agent as it improves the oxygenation by decreasing the right to left shunt as a result of increase in SVR.[9] Any decrease in SVR is treated with α-adrenergic agonists such as phenylephrine or norepinephrine and intravenous fluids and infundibular spasm is corrected with beta blockers such as propranolol or esmolol.[3] We used esmolol during induction and maintenance which helps to tackle tachycardia and spasm in infundibulam because of sympathetic stimulation..The operating room temperature should be kept at slightly higher side and adequate hydration has to be maintained with warm intravenous fluids because both hypothermia and hypovolemia contribute in increasing viscosity as well as right-to-left shunting. Neurosurgical requirements such as lax brain and maintenance of adequate cerebral perfusion pressure (CPP) are important with cautious use of diuretics such as mannitol, preventing dehydration. CPP are often maintained by optimising adequate systemic blood pressure. Adequate measures to preventair embolism such as preventing air bubbles in the intravenous lines is important as there is a high risk of paradoxical air embolism due to right to left shunt.[10]

Dexmedetomidine, an alpha-2 agonist, was used for the maintenance of an aesthesia because of its sedative, analgesic and central sympatholytic property. Dexmedetomidine inhibits the sympathetic system, balancing the cardiostimulatory effects of ketamine, thereby maintains a stable hemodynamic profile. Lower concentration should be used to avoid hypotension and bradycardia occuring with higher doses of dexmedetomidine. Volatile an aesthetics when administered in lower concentrations, improve arterial oxygenation by causing relaxation of the muscle spasm in RVOT and decrease the total body oxygen consumption. We did ABG before induction and after extubation as pulse oximetry is not are liable indicator of oxygenation due to severe cyanosis. We used multimodal analgesia to tackle pain as increased sympathetic activity due to pain may trigger cyanotic spell in the perioperative period.[11]

Even with favourable outcome, non-cardiac surgery still carries high-risk after operation. Observing them in high-dependency bed or intensive care perioperatively, warrants assurance of noticing and treating any adverse events like dysrrhythmia, cardiac ischaemia, dehydration, pain, ventilator issues, etc., preventing any further deterioration.[4,12]

CONCLUSION

Anaesthesia management of children with TOF presenting for non-cardiac surgery requires a thorough understanding of the pathophysiology of this condition and the altered haemodynamics.

A successful outcome in such a case required a meticulous preoperative assessment of the patient to assess the severity of the underlying heart disease, optimisation of the patient, carefully administered an aesthesia with meticulous planning, judicious use of drugs, along with strict monitoring, and vigilance with due considerations for neurosurgery.

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