Case Report

Anaesthetic Management for Brain Surgery in a Child with Uncorrected Tetralogy of Fallot in a Resource-Limited Setting

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Abstract

Tetratology of Fallot (TOF) is a rare congenital heart defect with Ventricular septal defect (VSD) as one of its four components. VSD predisposes this set of patients to developing complications of which brain abscess is one, due to shunting of blood from the right to left ventricle and concomitant loss of innate immunity of pulmonary filtering and removal of bacteria, clot and other organisms in blood from the right heart. Surgical evacuation of brain abscess in patients with background TOF could be challenging as administering anaesthesia could increase morbidity, mortality and pose great risk to this set of patients. We report a case of a six-year-old male child with an uncorrected TOF who developed multiple brain abscess and had surgical intervention (burr-hole and evacuation of abscesses) under general anaesthesia, and our challenges in a resource-limited hospital setting. A good anaesthetic plan, selection of appropriate anaesthetic medications to prevent right to left shunting of blood in the heart, using a pure alpha-receptor agonist; Phenylephrine, and excellent surgical intervention were major determinant of this patient survival without apparent neurological sequelae.

Keywords: Anesthesia, brain abscess, phenylephrine, tetralogy of Fallot

Introduction

Tetralogy of Fallot (TOF) is a congenital cardiac disease that consists of four defects, namely ventricular septal defect, pulmonary stenosis, right ventricular hypertrophy, and a large overriding aorta.[1] It is a rare condition; however, it is the most common cyanotic congenital cardiac disease with a prevalence of 5.1/10,000 children, as was reported in a hospital-based study, while its prevalence in congenital heart disease was 14.7% of all congenital heart diseases with a male predominance.[1] Few patients with TOF reach adulthood with an average life expectancy of 12 years if corrective surgery is not done.[2] Ojji *et al*. in Abuja, Nigeria, observed that the short lifespan in patients with uncorrected TOF is partly due to compensatory left ventricular hypertrophy and possibly systemic to pulmonary shunting via internal mammary arteries.[3]

The most common presentation is cyanosis and easy fatigability. Others include failure to thrive, congestive cardiac failure, and brain abscess.[2] Brain abscess is a focal infection of the brain parenchyma with resultant suppuration. It is common in adults and rare in the paediatric age group except in high-risk groups like children with congenital heart disease. In patients with TOF, cerebral abscess is one of the common presentation.[4]

Anaesthetic management in uncorrected TOF is often challenging, and the presence of brain abscess makes the patient high risk. In this article, we report a rare case of a six-year-old boy with uncorrected TOF who had emergency burr holes for drainage of left cerebral abscesses under general anesthesia.

Case Report

The patient was referred to our paediatric cardiology clinic on account of echocardiographic findings suggestive of TOF.

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He presented with a five-year history of easy fatigability, six-week history of fever and weakness of all limbs, and an episode of generalized tonic-clonic seizure four days before presentation.

Easy fatigability was noticed during late infancy, but there was no orthopnea, paroxysmal nocturnal dyspnea, or fainting spells. However, there was a positive history of squatting on exertion.

On examination, a small-for-age male schoolchild weighing 17 kg, sleeping but easily arousable, warm to touch (temperature: 37.8°C), he had central cyanosis with a Grade IV digital clubbing. He was not pale, anicteric and had no pedal edema.

The respiratory rate was 32/min, HR: 100 bpm, blood pressure: 100/60 mmHg, and SpO2: 77%–82% on room air. Chest auscultation revealed a systolic murmur loudest on the left sternal lower border.

The positive findings from the regional examinations were noted in the neurologic examination which revealed a fully conscious patient, but had right facial nerve palsy, global hypotonia, right hemiparesis and exaggerated deep tendon reflexes on the lower limbs. The findings in other aspects of his regional examination were normal.

An impression of TOF complicated by brain abscess to rule out cerebrovascular accident was made.

Cranial computed tomography scan [Figure 1] revealed multiple well-circumscribed hypodense lesions on the left cerebral hemisphere involving all the lobes with isodense covering and perilesional hypodensity. There was a significant midline shift and effacement of the left lateral ventricle. There was ring enhancement on contrast injection, and there was no evidence of bone involvement.

Brain magnetic resonance imaging [Figure 2] confirmed similar findings, all suggesting multiple left cerebral abscesses.

The packed cell volume was 55%. Serum electrolytes were all normal except serum potassium which was 2.1 mmol/l. His international normalized ratio was 1.1.

The patient was evaluated by the neurosurgical and anaesthetic teams and optimized for surgery by correction of dehydration using intravenous (IV) fluid normal saline 20 ml/kg and correction of hypokalemia with 20 mmol of iv potassium chloride via syringe driver over six hours. He was commenced on iv ceftriaxone, antipyretic (iv paracetamol). Prophylactic anticonvulsant (iv phenobarbital) was also included. He was assigned American Society of Anesthesiologists physical status Grade IV E, and planned for general anaesthesia with endotracheal intubation. Fasting was commenced while parenteral hydration was ensured.

Anaesthetic Management

In the operating room, standard monitoring was commenced using a multiparameter monitor. His baseline pulse rate was 110 bpm, blood pressure was 110/75mmHg, respiratory rate was 28/minute, SPO2 was 77-82%, however the SPO2 improved to 93-95% after preoxygenation with 100% oxygen by an oxygen delivering facemask. A Foley's catheter was inserted for urine output monitoring, and electrocardiogram monitoring was also commenced.

Anaesthesia was induced with 0.25 mg/kg of IV midazolam and 1.5 mg/kg of IV ketamine. Endotracheal intubation was facilitated with 0.1 mg/kg of IV pancuronium after positive test ventilation was confirmed. The patient was connected to an anaesthesia machine via a closed anaesthesia breathing circuit and anaesthesia was maintained with oxygen–sevoflurane mixture and mechanically ventilated at a tidal volume of 150 ml and 20 cycles per minute with a PEEP of 5 cmH2O. Analgesia was provided with IV pethidine 0.5 mg/kg and IV paracetamol 15 mg/kg. One gram of IV ceftriaxone was

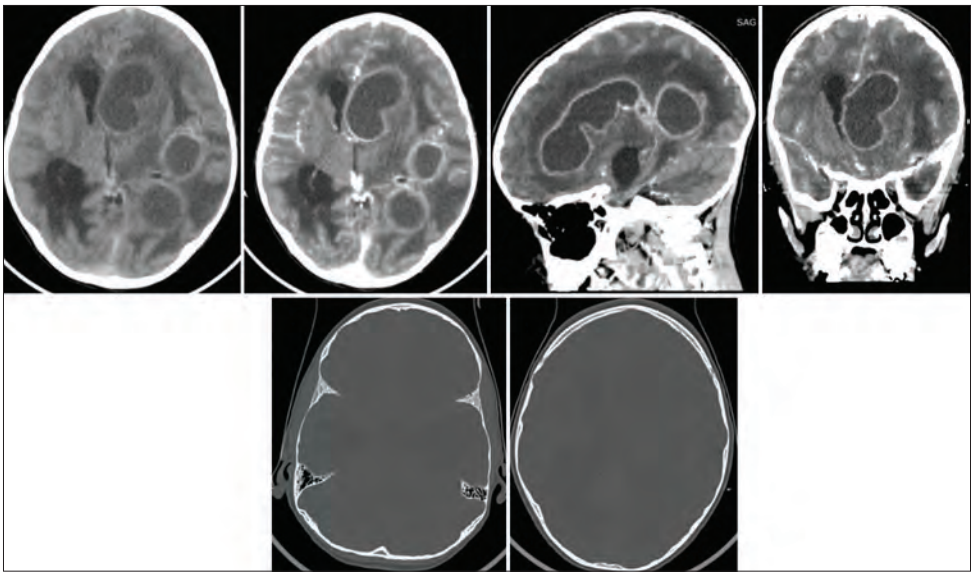

Figure 1: Cranial CT scan with contrast. CT: Computed tomography

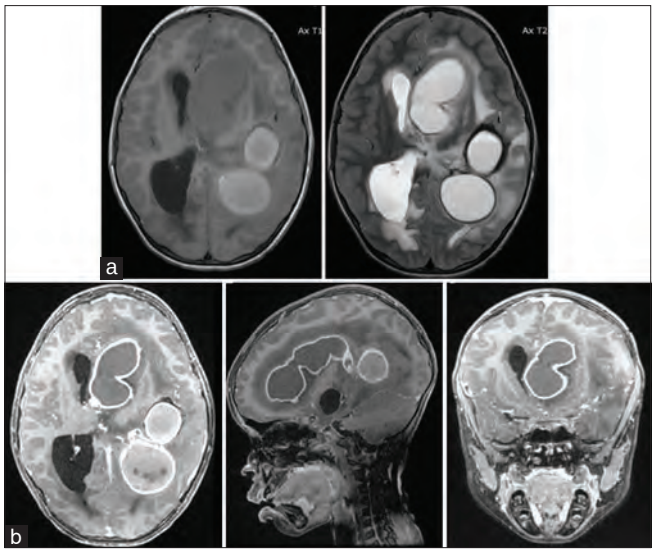

Figure 2: Brain MRI: (a) T1W and T2W, (b) T1 contrast-enhanced. MRI: Magnetic resonance imaging

administered, and a bolus of 20 ml/kg of Ringer's lactate was given to correct the fluid deficit and improve polycythemia, while maintenance fluid was delivered at 54 ml per hour.

Sevoflurane was at an MAC of 1.0 through most of the surgeries with a systolic blood pressure ranging from 130 to 140 and diastolic of 75–90 mmHg resulted to SpO2 of 90%–92% and PR >100 bpm. Shortly after commencement of surgery, hypotension with systolic blood pressure below 100mmHg and Diastolic blood pressure below 65mmHg accompanied with cold extremities and Spo2 below 80% were observed. This was managed using intermittent boluses of iv phenylephrine 5mcg/kg to maintain a systolic blood pressure above 130mmHg and diastolic blood pressure above 70mmHg. This intervention also helped to improve Spo2 to values greater than 90% with no significant changes in the pulse rate.

The child had left frontal and parietal burr holes with drainage of cerebral abscesses; the procedure lasted for 75 min, and 40 ml of pus was drained. The aspirate specimen was sent for microscopy, culture, and sensitivity (MCS). At the end of the surgery, sevoflurane was discontinued and residual neuromuscular blockade was reversed using IV neostigmine 0.05 mg/kg premixed with atropine 0.02 mg/kg. Extubation criteria were not met after this, and the patient was transferred to the intensive care unit (ICU) with an endotracheal tube *in situ* and manually ventilated with a bag-mask device connected to an oxygen source while being transported.

On admission into the ICU, the patient was noticed to have adequate respiratory effort and saturating at 90%, extubation was done after adequate suctioning of the oropharynx, and oxygen was administered via a partial rebreathing mask bag. Phenylephrine infusion was administered at 0.1 mcg/kg/min for another six hours till the child was hemodynamically stable. The antibiotic regimen was changed to meropenem and metronidazole empirically based on possible organisms and sensitivity patterns of cases of cerebral abscess encountered in the past while awaiting the result of the aspirate MCS. Analgesia, anticonvulsant prophylaxis, and IV fluids were continued. The patient was subsequently transferred to the neurosurgical ward for continued care after 24 h. Results of the MCS of aspirate did not contradict the already commenced antibiotics regimen, so the patient had the same IV antibiotics for four weeks and was discharged to the neurosurgery outpatient clinic for follow-up, and cardiothoracic outpatient clinic to be worked up for open-heart surgery and correction of TOF.

Discussion

In patients with TOF, bacteria from systemic circulation bypass the natural defense mechanism of the pulmonary circulation due to the intracardiac right-to-left shunt and are taken into the cerebral circulation unfiltered.[5,6] This hematogenous mode of spread accounts for the seed of the microorganism in multiple locations in the cerebral hemispheres.[5] Again, the chronic hypoxemia in the patient leads to polycythemia with consequent increase in blood viscosity.[5,6] This leads to foci of cerebral ischemia, which are easily colonized by microorganisms in the cerebral circulation. In spite of the above, some authors believe that the exact mechanism is unclear and yet to be unraveled.[5]

In the reported case, the patient had surgery for multiple left cerebral abscesses which was a complication of the uncorrected TOF which had earlier been diagnosed at infancy.

The major anaesthetic concern for this patient was preventing right-to-left shunting of the blood; therefore, measures to prevent hypotension, bradycardia, and dehydration were taken.

General anaesthesia with muscle paralysis and endotracheal intubation has been shown to be safe for patients with this condition and was the desirable anaesthesia technique for the procedure.[3]

Co-induction with ketamine and midazolam was the preferred induction approach, this is because ketamine increases peripheral vascular resistance preventing hypotension, and midazolam is cardiostable.[7] Propofol and thiopentone are unacceptable for induction of anaesthesia in this patient due to their cardio-depressant effect, a suitable alternative is co-induction with fentanyl-midazolam. Etomidate has also been used successfully for induction of anaesthesia in this group of patients, and dexmedetomidine is also a potentially useful drug.[7,8]

Sevoflurane was used for maintenance of anaesthesia. It is an excellent choice for inhalational anaesthesia as it has no cardio-depressant properties; however, a low MAC was used to prevent hypotension. Another inhalational agent that may have been used is isoflurane, while halothane is avoided to prevent worsening arrhythmia and a significant drop in peripheral vascular pressure.

Pancuronium was the muscle relaxant used for this procedure. It has vagolytic properties, though transient but desirable.

Suxamethonium was avoided due to its bradycardic effect which is undesirable. Other suitable muscle relaxants that could be used are vecuronium and rocuronium due to their cardio stability.

Adequate fluid hydration is also a very important aspect of this surgery as appropriate fluid management helps to reduce polycythemia, thereby reducing the risk of thromboembolic episodes.

A pure alpha-agonist phenylephrine was administered, and it is indicated in the treatment of hypercyanotic spells; an infusion of phenylephrine has been advocated by some authors from induction of anaesthesia.[8]

Conclusion

A selection of appropriate anaesthesia techniques and medications, use of a pure alpha-agonist like phenylephrine, adequate fluid hydration, and maintenance of a slightly high systolic and diastolic pressure are essential to reduce morbidity and mortality during surgery in patients with TOF. A good understanding of its pathophysiology is therefore important.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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