

Anaesthetic management of craniotomy for intracranial lesion in a child with uncorrected Tetralogy of Fallot

Nosheela Basit Rafique, Mohammad Hamid

Abstract

The case of a 16 years old female with uncorrected Tetralogy of Fallot, who operated for intracranial lesion in parieto frontal area with midline shift is presented. She had right ventricular hypertrophy, clubbing, central and peripheral cyanosis. Patient was anaesthetized keeping all measures required to avoid haemodynamic swings, tachycardia, desaturation, acidosis and dehydration. Pre-operative antibiotic cover was given to prevent bacterial endocarditis. Neurosurgeon, Paediatric cardiologist, Anaesthesiologist and Intensivist were involved in the preoperative planning and management of the patient. Haemodynamics were maintained and managed by monitoring continuous arterial line secured pre-induction and central line after induction. During surgery pain was controlled with fentanyl boluses intra-operatively and post operatively by tramadol infusion. Patient was extubated post operatively in the recovery room fulfilling the extubation criteria. She remained haemodynamically stable throughout the course. She was discharged on 5th post operative day from the hospital on SpO2 of 70-80% at room air.

Keywords: TOF, Intracranial lesion, Fentanyl boluses, Extubation criteria.

Introduction

Lesions of central nervous system(CNS) frequently occur in association with congenital malformations of the heart. The most serious CNS complications resulting from un-operated congenital heart disease are cerebral intravascular thrombosis, intracranial suppuration and cerebral abscess. The anatomic lesion which permits cerebral abscess formation in congenital heart disease (CHD) is a right to left shunt which allows blood to circulate in the arterial system without passing through the lungs. One of such defects are Tetralogy of Fallot (TOF) which typically comprises of four defects, i.e., ventricular septal defect (VSD), narrowing of Right ventricular outflow tract, over riding of aorta and right ventricular wall thickening. Brain abscess can develop

.....
Department of Anaesthesia, Aga Khan University Hospital, Karachi.

Correspondence: Nosheela Basit Rafique. Email: nosheela.rafique@aku.edu

due to bypassing the natural phagocytic action of lungs and allowing blood to directly pump into the systemic circulation. Persistently low arterial oxygen saturation manifests clinically as polycythemia, persistent cyanosis, clubbing of digits and poor exercise tolerance. The development of convulsions, focal neurologic abnormalities or clinical symptoms and signs of raised ICP are the alarming signs of presence of brain abscess. We are reporting the case of a young female who underwent neurosurgery for space occupying lesion in parieto-frontal region of the brain which was histopathologically confirmed as brain abscess.

Case report

A 16 year old female presented in Aga Khan University Hospital's Emergency department with the complaints of fever for two days and generalized tonic clonic seizures for half an hour. The patient had been diagnosed to have a space occupying lesion in the brain and was advised surgery three months ago. Her examination showed pulse:97/min; BP: 135/81mmHg; RR:20/min; SpO2:89-92% with FiO2 at 2L/min; ECG showed normal sinus rhythm with tachycardia, T-wave inversions in lead III and V1, strain pattern in anterior chest leads, right ventricular hypertrophy; Clubbing in both hands, mild peripheral and central cyanosis with pale complexion. She was a known case of uncorrected TOF. Her initial laboratory workup showed increased haemoglobin and haematocrit along with the following ABGs:7.41/65/23/14.9/-7.2. MRI brain revealed space occupying lesion at parieto-frontal region of brain with midline shift. Therefore,craniotomy for excision of the lesion was planned by the neurosurgeon. Neurosurgeon, Paediatric cardiologist, Anaesthesiologist and Intensivist were all involved in the preoperative planning and management of the patient. She was shifted to the operating room with 4L/minute of O2. OR preparation included bolus syringes of phenylephrine and epinephrine along with epinephrine infusion. In addition, Bicarbonate was also available in the room. Arterial line was secured before induction. Patient was induced with thiopentone sodium (4mg/kg), Fentanyl (2microgram/kg) and Atracurium (0.5mg/kg). Pre-operative antibiotics Cefazoline 1Gm and Vancomycin

500mg I/V was given after induction. Anaesthesia was maintained by isoflurane, fentanyl, oxygen and regular doses of atracurium boluses. Patient was monitored with pulse oximetry, ECG, real time blood pressure via arterial catheter in right radial artery and was well hydrated by maintaining CVP >12mmHg. Surgery lasted for 4hours and patient remained haemodynamically stable and did not require vasopressors or inotropes. Saturation was maintained around 78%-81% at 100% FiO₂. Since patient had VSD with right to left shunt so all the intravenous lines were kept air free to prevent paradoxical air embolism. Intravenous Dexamethasone 8mg was given intra operatively on surgeon's request. One unit of packed red blood cells was transfused in the middle of surgery and Phenytoin 500mg infusion started at slow rate towards the end of surgery.

Patient was shifted to recovery room and intubated for the concern of decreased sensorium in response to Phenytoin infusion. She was reversed from neuro-muscular blocker (Atracurium) and extubated once optimum GCS (9/10) was achieved and was obeying commands. She remained stable and was shifted to special care unit (SCU) and then to the ward on the next day. Saturations remained between 70- 85% at 5L/min of O₂. Chest physiotherapy and spirometry was done to prevent postoperative pulmonary complications. Pain management was done with tramadol infusion. She was discharged on 5th post-operative day from the hospital on SpO₂ of 70-80% at room air.

Discussion

Brain abscess is a serious, life-threatening condition which requires urgent surgery. Severe neurological deficits have been found in patients who survived brain abscess. It is a focal intracerebral infection that begins as a localized area of cerebritis and develops into a collection of pus surrounded by a well-vascularised capsule.^{1,2} Solitary brain abscesses are usually caused by local spread of infection from adjacent structures such as the paranasal sinuses, the middle ear or from infected traumatic or surgical wounds. Additionally, haematogenous spread from a distant focus such as the heart or lung can also seed the infected material.

In a study, CHD remained the commonest underlying cause of brain abscess among children.³ Congenital heart defects are the most common inborn defects and occur approximately in 0.8% of the new born infants.⁴ Out of these about 5% of the newborns have TOF.⁵ Correlation between the TOF and brain abscess is still unclear, most commonly found in the supratentorial compartment. In few older studies, underlying cardiac

condition was seen as the only cause of 18.7% of all brain abscesses.⁶ Kagara et al had reported 4% cardiac origin brain abscesses found in the posterior fossa. These were all located in the cerebellum.⁷

Patients with cyanotic CHD may develop minute encephalomalacia due to severe hypoxaemia and increased blood viscosity resulting from compensatory polycythemia. The increased blood viscosity and reduced blood flow in the microcirculation may induce cerebral thrombosis or exaggerate minute encephalomalacia during dehydration or cardiac dysfunction, and shunted blood containing infectious organisms at such sites may be followed by focal cerebritis.

During the procedure hypoxia, hypercarbia, hypothermia, hypovolemia and acidosis was avoided to prevent high pulmonary vascular resistance which may lead to right to left shunt. All the IV lines were kept air free to prevent air embolism, and N₂O was avoided throughout the procedure (It may increase air bubble size). CVP in right internal jugular vein was secured post induction to guide fluid therapy and also to remove air in case of air embolism during surgery. Preoperative antibiotic cover was also given to prevent bacterial endocarditis.

Perioperative haemodynamic stabilization was the biggest concern for the clinicians and for the safety of the patient. Therefore a paediatric cardiologist and intensivist were included in the planning and post operative care. Myocardial depressant drugs were avoided during the procedure, and inhalational anaesthetic (Isoflurane) was given at 1-1.5% with 100% O₂. At the same time systemic venous resistance (SVR) was maintained by using intravenous phenylephrine boluses. (As, reduction in SVR increases right to left shunt). In addition patient was kept adequately hydrated in order to avoid hypotension. Blood loss during the procedure was 450ml which was replaced with equal amount of packed cells. Urine output was maintained approximately at 1ml/kg/hr.

The patient was subjected to respiratory alkalosis by hyperventilating intentionally during the procedure, as it is helpful in reducing intracranial pressure and prevents pulmonary vasoconstriction. Patient was kept normothermic (36-36.8°C) by giving humidified breathing gases; using fluid warmer for all I/V fluids and using K-thermia blanket under the patient as hypothermia increases pulmonary vascular resistance.

Pain is also a very important component particularly

in the postoperative period. Pain during the surgery causes sympathetic discharge leading to greater right ventricular outflow tract obstruction. Inadequate pain relief can also result in ineffective chest physiotherapy leading to pneumonia and difficulty in ambulation. In our case we kept our patient pain free with fentanyl boluses intraoperatively and later post operatively pain was controlled by tramadol infusion.

With the help of surgical intervention towards the end of the 19th century and the introduction of antibiotic therapy in the mid-20th century the mortality and morbidity of invariably fatal brain abscess has considerably reduced. There have been a number of more recent advancements towards the management of patients with brain abscesses which included early computerised tomography (CT scan), refinements in neurosurgical technique, timely use of proper antimicrobial agents has also contributed to the better prognosis of these patients.¹ Early diagnosis and proper management of congenital and rheumatic heart disease also contribute towards the better survival.⁸

Brain abscess in patients with CHD constitutes 11% of all brain abscesses and the incidence is decreasing in developed countries due to early surgical interventions in patients with congenital heart diseases.⁹ The prognosis for cardiogenic brain abscess is poor and mortality rate ranges from 30% to 71%^{7,10} even with available surgical treatment.

Conclusion

The anaesthetic management of uncorrected TOF patient admitted for high risk intracranial surgery was presented. Management included pre-operative planning and communication with involved disciplines, avoidance of pulmonary hypertension maintenance of systemic vascular resistance, air-free intravenous lines and post-operative pain management. As such cases are extremely challenging and mortality associated, a multidisciplinary approach should be applied.

References

1. Mathisen GE, Johnson JP. Brain abscess. *Clin Infect Dis* 1997; 25: 763-81.
2. Kao PT, Tseng HK, Liu CP, Su SC, Lee CM. Brain abscess: clinical analysis of 53 cases. *J Microbiol Immunol Infect* 2003; 36: 129-36
3. Goodkin HP, Harper MB, and Pomeroy SL. Intracerebral abscess in children: historical trends at children's hospital Boston, *Pediatrics* 2004; 113: 1765-70.
4. Mohindra R, Beebe DS, Belani KG. Anaesthetic management of patients with congenital heart disease presenting for non-cardiac surgery. *Ann Card Anaesth* 2002; 5: 15-24.
5. Lovell AT. Anaesthetic implications of grown-up congenital heart disease. *Br J Anaesth* 2004; 93: 129-39.
6. Ghosh S, Chandy MJ, Abraham J. Brain abscess and congenital heart disease. *J Ind Assoc* 1990; 88: 312-4.
7. Kagawa M, Takeshita M, Yato S, Kitamura K. Brain abscess in congenital cyanotic heart disease. *J Neurosurg* 1983; 58: 913-7.
8. Char G, West K, Jaggon J. Intracranial Abscesses: Epidemiological trend over a 39- year period at the University of the West Indies. *Int J Third World Med* 2009; 8: DOI: 5580/27.
9. Mamoalam TJ, Rosenblum ML. Trends in the management of bacterial brain abscesses: a review of 102 cases over 17 years. *Neurosurg* 1988; 23: 451-8.
10. NEWTON EJ. Haematogenous brain abscess in cyanotic congenital heart disease. *Q J Med* 1956; 25: 201-20.