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A case of uncorrected D-TGA for craniotomy in cerebral abscess: anaesthesia management

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Key points

Congenital cardiac anomalies pose many challenges during anesthesia due to anatomic and physiological alterations. The inherent complications associated with these disorders necessitate vigilance for providing anesthesia to even seemingly simple surgical intervention.

Abstract

Transposition of Great Arteries (D-TGA) is one of the most common cyanotic congenital heart defect in newborn, having atrioventricular concordance with ventriculoarterial discordance where Aorta arises from right ventricle and pulmonary artery from left ventricle. Patients with cyanotic congenital heart disease (cCHD) are prone to develop frequent brain abscesses. Mortality rate remains very high in these patients. Anesthetizing children with cCHD and a brain abscess necessitates use of an anesthesia regimen appropriate to both cCHD and intracranial surgery. Here, we share our experience of anesthesia management of uncorrected Dextro Transposition of Great Arteries (D TGA) for craniotomy and intracerebral abscess drainage.

Keywords: Dextro transposition of great arteries; cerebral abscess

Introduction

Brain abscess is an uncommon and life threatening intracranial infection characterised by purulence and inflammation in one or more localised areas within the brain parenchyma. It results from spread of infection from surrounding non-neuronal tissues eg middle ear, fracture skull. Intracranial surgery, hematogenous spread as in congenital heart disease with a right to left shunt (5-18.7%) or a direct introduction into the brain.[1] (D-TGA) accounts approximately 5% to 7% of all congenital heart diseases. There is discordance of the ventriculoarterial connection. Areas of mixing of oxygenated and non oxygenated blood are vital for the survival of the baby. Without mixing, the two circuits remain separate, leading to death from systemic hypoxaemia and acidosis. The possible locations for mixing are via a patent foramen ovale (PFO), atrial septal defect (ASD), ventricular septal defect (VSD), patent ductus arteriosus (PDA), or through bronchopulmonary collaterals. The larger size of the communications between chambers leads to more mixing of oxygenated and deoxygenated blood resulting in higher oxygen saturation and better heamodynamic stability.[2]

This intercirculatory mixing of blood leads to hypoxia and its consequent polycythemia and hyperviscosity. The latter results in sluggish blood flow in cerebral microcirculation, microthrombi formation and direct entry of organisms, emboli, infected seed to cerebral circulation forming cerebral abscess. Also, abscess related problems like vomiting, dehydration, acid base and elec-

trolyte imbalance, raised intracranial pressure (ICP), seizures, make them high-risk candidates for abscess excision under GA.[2]There is a paucity of anesthesia literature on the coexistence of these two conditions.We report a case of 6 years old child of TGA with cerebral abscess for drainage.

Case report

A 6 years old male child weighing 12 kg, diagnosed case of a D-TGA presented with fever, vomiting and generalized seizures since 20 days. General examination revealed an afebrile patient, central cyanosis with on air O_2 saturation (SpO_2) of 71%, grade 4 clubbing of digits in all four limbs,no peripheral signs of infective endocarditis. An ejection systolic murmur in pulmonary area was heard in cardiac examination. Neurologically he was irritable with Glasgow coma scale (GCS) of 13(E4M5V4). An upper motor neuron type of facial palsy with paresis of right side of body was noted. Kernig's sign was negative. Laboratory investigations showed Hb-15.4g% with Hct-43.9%, platelets counts-137,000/cmm, total leucocyte count-14500/cmm with normal liver and renal function tests. Arterial blood gas revealed PO_2 40.6mmHg and SO_2-72% without acidosis. Computed tomography (CT) Brain scan showed cystic enhancing lesion in the left temporoparietal region, with perilesional oedema causing midline shift (Figure 1). X ray chest showed an egg shaped heart with hyperaemic lung fields. 2D echo was suggestive of congenital heart disease, d-TGA, large OS ASD 20mm with bidirectional shunt, dilated Right atrium and Right ventricle with severe Pulmonary Arterial Hypertension (PAH). He was receiving antibiotics and antiemetics intravenously. Inotropes/vasopressors, prophylactic antibiotics, anticonvulsant, anti edema drugs, Fresh Frozen plasma and Platelets were kept ready prior to procedure. After obtaining informed consent from parents, confirming starvation, child was taken in the theatre. After preoxygenation for three min SaO_2 increased to 76%. Care was taken to avoid intravascular air. After intravenous premedication with + injection Glycopyrrolate 0.004 mg/kg, injection midazolam 0.02 mg/kg anaesthesia induction was carried out with injection fentanyl 2 µg/kg + ketamine 1 mg/kg vecuronium 0.1 mg/kg and trachea was intubated.

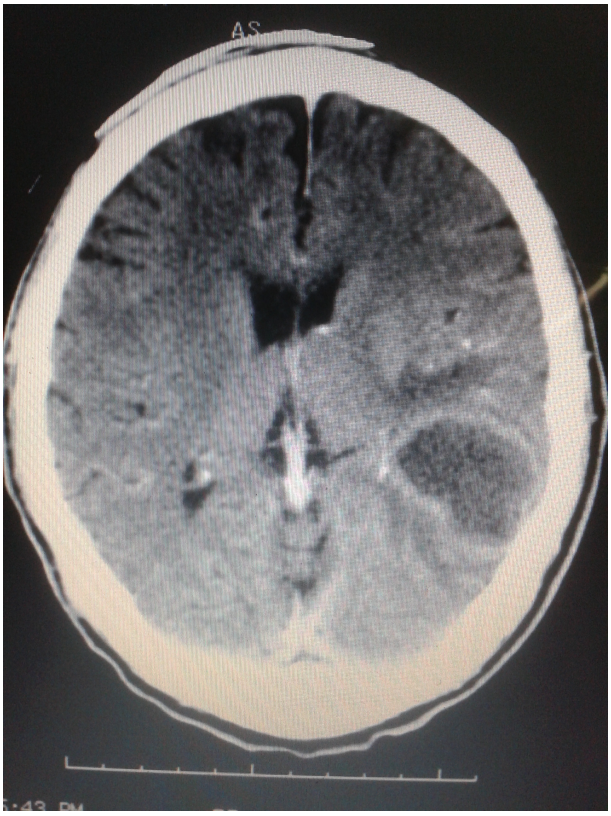

Fig. 1. Computed tomography (CT) Brain scan showed cystic enhancing lesion

Right Internal jugular vein cannulation was done using 5.5 Fr triple lumen catheter. Anaesthesia was maintained on O_2 (50%) + Air (50%) + 1-2% sevoflurane + inj fentanyl 2 µg/kg/hr and inj vecuronium infusion 0.08mg/kg/hr with controlled ventilation. Intraoperative monitoring included Electrocardiogram, Pulse Oximetry, $ETCO_2$, Non invasive blood pressure (NIBP), central venous pressure (CVP), and arterial blood gases. Intraoperatively Ringer lactate was infused (10 ml/kg/hr) to maintain CVP 6 – 8 cm of H_2O, IV Mannitol 2 ml/kg was given to reduce cerebral oedema. The surgery lasted for about two hours and throughout the procedure there was no episode of hypercyanotic spell or hypoxia. The heamodynamics remained stable with HR 96 to 114/min, BP 80to100 mmHg systolic, SaO_2

ranging between 82–86% and $PaCO_2$ between 30–33 mmHg. Craniotomy with drainage of encapsulated abscess cavity was performed by Neurosurgeon. At the end of surgery, residual neuromuscular blockade was reversed, patient extubated once awake and monitored in Neurosurgery ICU for 24 hrs postoperatively. IV Paracetamol 15 mg /kg body weight 8 hourly was used for postoperative analgesia.

Discussion

Patients with cyanotic congenital heart disease (cCHD) are prone to develop frequent brain abscesses. TGA if uncorrected, has a 30% mortality rate in the first week of life, 45% in the first month and 90% in the first year. Those who survive this period present with the problems of preexisting hypoxia and cyanosis, polycythemia, hypercoagulability, thrombotic complications, coagulopathies. They have higher incidence of systemic infections due to bypass of the filtering of pulmonary capillaries leading to brain abscess.[2] Treatment of brain abscess is with systemic antibiotics. However, abscesses which are larger than 2 cms in diameter, causing midline shift, multiple abscesses require surgical excision under general anaesthesia in small children.[3,4] Mortality rate remains as high as 13% in patients of cyanotic congenital heart disease (cCHD) with brain abscess[4]. The risk further increases under anaesthesia due to presence of other extracardiac malformations (20-45%)[4], difficult airway, a fragile cardiopulmonary status and various systemic and coagulation complications, together with abscess-induced problems such as raised intracranial pressure (ICP), seizures, dehydration, electrolyte imbalance. Anesthetizing children with cCHD and a brain abscess necessitates use of an anesthesia regimen appropriate to both cCHD and intracranial surgery. Intraoperative goals include maintenance of hemodynamic stability and oxygenation and prevention of cyanotic spells and arrhythmias. Perioperative hypoxia leads to hyperviscosity of blood and coagulation abnormality requiring phlebotomy if haematocrit is above 65% . If coagulation abnormalities are present, platelet concentrates are needed. In our patient the PCV was 43.9%, and did not have deranged coagulation profile. The amount of shunting of blood in dTGA is determined by the ratio of the systemic vascular resistance (SVR) to pulmonary vascular resistance (PVR). As SVR is increased, right to left shunting decreases. However increase in PVR leads to abrupt worsening of cyanosis, tachycardia, hypotension. Hence, one should maintain systemic vascular resistance, minimize pulmonary vascular resistance,maintain cardiovascular stability. The optimal induction regimen for general anaesthesia in cCHD should aim to improve arterial blood oxygen saturation (SaO_2)[5]. Induction of anaesthesia increases arterial saturation probably due to high oxygen concentration plus decreased oxygen consumption with induction of anaesthesia and muscle paralysis[6]. However, if systemic vasodilatation occurs, it may exacerbate the right to left shunting and intensify hypoxia. We used a combination of Fentanyl and ketamine in our case for induction of anaesthesia which actually improved arterial oxygenation and maintained it between 82-86% intraoperatively. Ketamine decreases the right to left shunt by increasing systemic vascular resistance[4,7,8,9] and is a suitable induction agent in such cases. Hypotension, hypovolemia, acidosis, hypoxia, and hypercarbia increase intraoperative shunting and should be avoided. Hypocarbia and diuretic therapy are usually employed to obtain lax brain. Diuretic should be used carefully to avoid hypovolemia and reduced right ventricular output. Mannitol in a small dose is ideal as it reduces blood viscosity also. We avoided hypovolemia by maintenance of intravascular volume[7]. Early extubation is preferred to prevent further reduction in pulmonary blood flow due to prolonged ventilation. Adequate post operative analgesia is essential as pain increases pulmonaryvascular resistance and sudden increase in oxygen demand.

Conclusion

Brain abscess is a known complication in patients with cyanotic congenital heart disease, which must be diagnosed early and treated aggressively. In patients with

un-operated cyanotic congenital heart disease, early corrective surgery or palliative shunts i-e Blalock Taussig, Bidirectional Glen shunt for these cardiac malformations would be a definitive way of reducing this complication. A carefully administered GA with controlled ventilation, maintaining cardiac output, normal sinus rhythm and keeping PVR relatively lower than SVR along with lax brain is recommended.

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