Case Report

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Anaesthetic Management of Intracranial Abscess in a Patient with Surgically Uncorrected Double Outlet Right Ventricle with Transposition of Great Arteries

Anaesthesia Section

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ABSTRACT

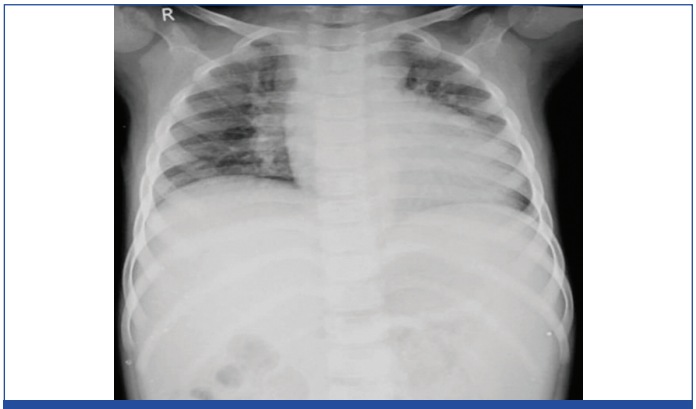
Double Outlet Right Ventricle (DORV) with Transposition of Great Arteries (TGA) is a rare variant. The ventriculoarterial discordance due to this anatomical defect leads to parallel circulation. The postnatal survival depends on mixing of oxygenated and deoxygenated blood at various levels by defects like Atrial Septal Defect (ASD), Ventricular Septal Defect (VSD), Patent Ductus Arteriosus (PDA). Anaesthetising children with cyanotic Congenital Heart Disease (cCHD) and brain abscess necessitates use of anaesthesia protocol appropriate to both cCHD and intracranial surgery. Anaesthetic considerations related are maintenance of intravascular volume and preload, avoidance of precursors to acidosis such as hypothermia, hypercarbia, and hypotension and challenge for anaesthesiologist is to maintain Pulmonary Vascular Resistance (PVR) and Systemic Vascular Resistance (SVR) by pharmacological and ventilatory means. This report is about a seven-year-old child who was posted for emergency craniotomy and abscess drainage, after routine preanaesthetic check-up. Induced with fentanyl, ketamine and atracurium, anaesthesia was maintained on oxygen, air and sevoflurane. Intraoperatively, normothermia was maintained, and blood pressure was maintained by noradrenaline infusion, metabolic acidosis was corrected with soda-bicarbonate. In between the procedure, the patient had an episode of Supraventricular Tachycardia (SVT), which was managed with lidocaine, metoprolal and adenosine. In such cases, it is vital to pay attention to any potential anaesthetic interactions.

Keywords: Congenital heart disease, Neuroanaesthesia, Paediatric anaesthesia

CASE REPORT

A seven-year-old, 20 kg female child, was brought by her parents to the emergency department with complaints of convulsions for five days, a right-sided weakness for five days, and numbness sensation over the right arm followed by convulsions which were focally involved upto the right arm, fever since one day. The mother gave a history of CHD (unspecified), diagnosed at three months of age, when the child presented with failure to thrive and bluish discolouration of the body on crying. She also gave a history of similar complaints in the year 2020 and was diagnosed with a tubercular brain abscess and operated on for the same. Postsurgery, antitubercular treatment was started and discontinued by parents themselves one month later without consulting doctors, with no further follow-up. After the primary assessment, routine blood investigations were sent, Magnetic Resonance Imaging (MRI) brain and 2D echo were done and then patient was admitted to the paediatric Intensive Care Unit (ICU) and treated with syrup. levetiracetam 20 mg/kg/day, syrup sodium valproate 10 mg/kg/day, injection (inj.) paracetamol 15 mg/kg IV. Patient was diagnosed with left frontoparietal-temporal abscess with DORV with TGA. She was posted for craniotomy and abscess drainage on emergency basis.

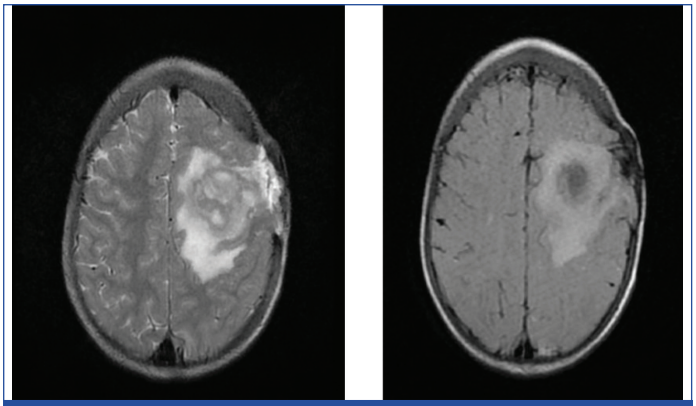
Her lab investigations showed haemoglobin 14.8 gm/dL, haematocrit 49.2%, Total Leukocyte Count (TLC) and platelet count, coagulation profile, and other laboratory investigations were within normal limits. A chest radiograph showed increased pulmonary vascular markings [Table/Fig-1]. On preanaesthetic assessment, the child fell under category 4 of the American Society of Anaesthesiology (ASA) grading [1]. On examination, central cyanosis and clubbing grade 2 was noticed. Respiratory system examination showed bilateral equal air entry, with a saturation of 55% on room air Respiratory Rate (RR) of 22 breaths/minutes. An airway assessment was done and was adequate. A single loud second heart sound and an audible pan systolic murmur were noticed during a cardiovascular system evaluation. Central nervous system evaluation revealed that the patient was cognizant and had a Glasgow Coma Score (GCS) of 15, hypotonia and limb power score of 0/5 for the right upper limb, 3/5 for the right lower limb, and 4/5 for the left upper limb and lower limb.



[Table/Fig-1]: Chest radiograph showing increased pulmonary vascular markings.

A 2D Echo revealed a DORV with malposition of great vessels, the aorta was right and anterior, pulmonary artery was left and posterior, large inlet VSD with bidirectional shunt, slight left to right shunt ASD, severe valvular, subvalvular pulmonary stenosis with pressure gradient 64 mmHg, confluent pulmonary artery stenosis (right and left)=9.5 mm.

MRI brain contrast revealed a large thick-walled multiseptated peripheral enhancing lesion in the left high frontal-parietal lobe measuring around 2.1×3.2×2.9 cm (CC×TR×AP) with associated perilesional oedema. Mass effect was seen in the form of effacement of adjacent sulci. The lesion was heterogeneous, hyperintense on T2WI, and suggestive of residual/recurrent tubercular abscess. ([Table/Fig-2a]-T2WI, [Table/Fig-2b]- FLAIR).



[Table/Fig-2]: T2 Weighted Image (T2WI) and FLAIR image: Large thick-walled multiseptated peripheral enhancing lesion in the left high frontal-parietal lobe measuring around 2.1×3.2×2.9 cm (CC×TR×AP) with associated perilesional oedema. The lesion was heterogeneous, hyperintense and suggestive of residual/recurrent abscess. (Images from left to right)

The patient was taken for the procedure after the parents were informed about the significant risk. Standard anaesthetic monitoring devices were attached. Intravenous access was carefully secured with a 22-G after flushing it off air bubbles. Preoperatively patient was afebrile with a Heart Rate (HR) of 110 beats/min, Blood Pressure (BP) of 70/50 mm Hg, and SpO_2 of 57% on room air. Thirty minutes before giving the skin incision, ceftriaxone 50 mg/kg was administered intravenously. Preoxygenation was given over four minutes, and the rise of SpO_2 to 70% maximum was noticed. Fentanyl 2 mcg/kg was given as premedication. Ketamine 2 mg/kg and atracurium 0.5 mg/kg was given as induction agent and muscle relaxant, respectively. The patient was intubated with a 5.5-cuffed endotracheal tube. Sevoflurane in 100% oxygen with MAC ranging from 0.8 to 0.9, and atracurium top-ups were used to maintain anaesthesia. The patient was placed on volume control mode with a tidal volume of 8 mL/kg, RR of 28 breaths/min, without Positive End-Expiratory Pressure (PEEP). Invasive temperature monitoring was done throughout the procedure. Postintubation, vitals are stable with SpO_2 of 60%. Ultrasound-guided 5.5 fr triple lumen central venous catheter was secured in the right internal jugular vein. A 22G peripheral catheter was secured in the right radial artery for invasive blood pressure monitoring. Noradrenaline infusion was started because of low blood pressure (70/40 mm Hg) half an hour after incision at the rate of 0.5 mcg/kg/min and titrated according to blood pressure. Arterial Blood Gas (ABG) analysis was done immediately after intubation and showed uncompensated metabolic acidosis with hypoxaemia. The sodiumbicarbonate correction was given accordingly. Mannitol 1.0 gm/kg, vancomycin 15 mg/kg/dose, and levetiracetam 20 mg/kg were given intraoperatively. Throughout the procedure, SpO_2 was in the range of 50-66%. End-tidal carbon dioxide ($EtCO_2$) was maintained between 30 and 38 mm Hg. Two hours after correction, ABG was repeated, which indicated hypoxaemia.

In between the procedure, the patient had an episode of narrow QRS complexes with absent P waves SVT with HR of 181 beats/min, SpO_2 of 50% and BP of 80/50 mm Hg, $EtCO_2$=38 mm Hg, the temperature of 36.6ºcelsius, which was treated with i.v. lidocaine 20mg stat followed by metoprolol 0.5+0.5 mg, due to non availability of adenosine at that moment, there was drop in HR from 181 to 138 beats/minutes, but rhythm did not revert. An injection adenosine was arranged in a short period, and 2 mg (0.1 mg/kg) was given rapidly as a bolus via the central line, followed by a saline flush; rhythm was reverted back to sinus rhythm with HR of 104 beats/min. A defibrillator was kept on standby to give synchronised cardioversion if needed, and diltiazem was also made available. ABG was repeated at the event's time, which showed persistent hypoxaemia. Serum electrolytes (sodium, potassium, calcium, magnesium) were within normal limits. Later, the procedure went uneventfully.

Towards the end of the procedure, all anaesthetic agents were stopped; paracetamol 15 mg/kg was given intravenously for analgesia, neostigmine and glycopyrrolate were given to reverse the neuromuscular blockade, and after the return of adequate muscle power and reflexes, the patient was extubated. Postextubation ABG was done which showed hypoxaemia. Overall, the surgery took four hours. Fluid management was done according to the Holliday Segar formula with crystalloids. Urine output was 1.5-2 mL/hour. Blood loss of 160 mL was noted, which was replaced with 80 mL blood. Normothermia was maintained all over the procedure. After stable vitals, the child was transferred to the Paediatric Intensive Care Unit (PICU). Postoperatively, the patient was vitally stable with no episodes of SVT, iv paracetomal was used as analgesic, antibiotics and antiepileptics were started accordingly, and the child was discharged on a postoperative day 10 after paediatric cardiologist's/consultant opinion.

DISCUSSION

The DORV may exist as a single condition or in association with cardiac or extracardiac anomalies. The incidence ranges from 0.03 to 0.14 per 1000 live births. It occurs in about 1% of all CHD [2]. DORV with TGA accounts for 26% of cases of DORV [3]. According to the congenital heart surgery taxonomy and database, DORV was a spectrum of CHD in which both great arteries emerge fully or predominately from the right ventricle [4]. Patients with cyanotic cCHD are prone to develop cerebral abscesses. Those who survive infant period present with the problems of pre-existing hypoxia, hypoxaemia, cyanosis, hypercoagulability, polycythaemia, thrombotic complications, coagulopathies, ventricular dysfunction and possibility of infective endocarditis [5,6]. Prolonged preoperative fasting needs to be avoided and adequate hydration maintained to prevent hyperviscosity. i.v. cannula and all lines should be flushed appropriately to avoid air bubbles, as these patients are at increased risk of paradoxical air embolism due to bi-directional shunts.

Anaesthetic considerations are- maintenance of intravascular volume and preload and avoidance of precursors to acidosis such as hypothermia, hypercarbia, and hypotension [6]. In this case, hypovolemia was avoided by maintenance of intravascular volume hypotension was corrected with help of noradrenaline infusion and acidosis with Sodium bicarbonate correction. The PVR to SVR ratio affects SpO_2. A decrease in PVR increases pulmonary blood flow to peripheral alveoli, leading to higher arterial saturation. The task for anaesthesiologists was to regulate PVR and SVR via pharmacological and ventilator methods [7]. Fentanyl was chosen, as it causes the least amount of haemodynamic variation and minimises responsiveness of the pulmonary vasculature and provide adequate analgesia [7]. Ketamine was used because it is a potent, safe, rapid-onset agent that increases SVR there by decreases right to left shunt [8,9]. Sevoflurane was used in anaesthesia maintenance, while noting that the systemic blood flow to pulmonary blood flow ratio remains unaltered and systolic function of the left ventricle was maintained at 0.8-0.9 MAC of sevoflurane in 100% oxygen [10].

SVT was the most common dysrhythmia in children, with an estimated prevalence of 1 in 250 to 1000 paediatric patients [11]. Treating SVT in a patient who was undergoing surgery, necessitates a thorough yet quick analysis of possible causes. Potential causes include electrolyte imbalances, acidosis, hypercarbia, hypotension, hypoxaemia, hypothermia, mechanical irritation, and adrenergic stimulation brought on by the light plane of anaesthesia [12,13]. The cornerstone of SVT treatment was ventricular rate regulation. Na^+ channel blocker lidocaine and the beta-1 blocker metoprolol were used to regulate the rate [12]. Adenosine, effectively stops sinus node re-entry tachycardia that use the AV node as a component of the re-entrant mechanism [13]. In this case, adenosine reversed the sinus rhythm.

CONCLUSION(S)

The current case demonstrates that with a thorough understanding of pathophysiology and proper planning to avert potential complications, patients with complex CHD can be successfully anaesthetised for non cardiac surgery without incident.

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