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Case Report

Anaesthetic considerations in congenital single ventricle cyanotic heart disease patients undergoing non – cardiac surgery

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ABSTRACT

Congenital heart disease (CHD) is one of the most prevalent congenital disorders, with an incidence rate of 9.1 per 1000 live births. Lesions featuring single ventricle physiology make up about 20% of these cases. This condition involves an abnormal parallel circulation, leading to complications such as ventricular dysfunction, chronic hypoxia, polycythaemia, and infective endocarditis, which collectively increase mortality risk.[1] Patients with a single functioning ventricle have limited tolerance for changes in preload, afterload, myocardial depression, and fluctuations in pulmonary and systemic vascular resistance. Factors like reduced tidal volume, atelectasis, interstitial lung water, and hypoxic pulmonary vasoconstriction contribute to a balanced circulation, but these factors can be significantly altered during induction and intubation. Here, we report the successful management of a male child with single ventricle physiology who underwent an emergency burr hole procedure and tapping of a brain abscess under general anesthesia.

1. Introduction

A single ventricle is a rare occurrence in embryonic development, leading to the anatomical or functional loss of a ventricular chamber. Single ventricle physiology refers to a group of congenital heart defects characterized by having only one functioning ventricle.[2] Single ventricle physiology leads to admixture of arterial
and venous blood and mixed blood get distributed to both pulmonary and systemic circulqtion. Patients with single ventricle physiology present significant challenges to anaesthesiologist's during both cardiac and non-cardiac surgeries. The abnormal parallel circulation, coupled with ventricular dysfunction, chronic hypoxia, polycythaemia, and infective endocarditis, increases mortality risk.[3] We report on a patient with single ventricle physiology who underwent non-cardiac surgery.

2. Case Report

A 16-year-old boy weighing 40 kg presented to the emergency department with a one-week history of intermittent fever and headache, as well as two months of easy fatigability and exertional dyspnoea (Grade II). He had been diagnosed with congenital cyanotic heart disease since birth but had not received any treatment due to being asymptomatic. There was a history of failure to thrive. On examination, the child was fully conscious, oriented, and afebrile, though he appeared dull. An ejection systolic murmur was audible in the pulmonary area, and room air oxygen saturation was 93%.

Laboratory investigations revealed a haemoglobin level of 15.1 g/dl, with normal total leukocyte count, platelet count, coagulation profile, and other lab results.

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The ECG revealed a broad, bifid P wave in all limb leads and in lead V1, sinus bradycardia, and short PR interval.

2D Echo revealed poor echo window, double inlet single ventricle physiology, LVEF 56%, Hypoplastic Right Ventricle, moderate TR, moderate MR, mild-mod PHT, mild-mod AR, Dilated main pulmonary artery & PDA with shunt.

CT scan of brain revealed well defined smooth walled peripherally enhancing hypodense collection in right Frontal-parietal temporal lobes. Features suggestive of brain abscess with adjacent focal meningeal inflammation. Emergency burr hole and tapping of abscess was planned under general anaesthesia.[4]

3. Intraoperative Management

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. Preoperatively patient was afebrile with a Heart Rate (HR) of 68 beats/min, Blood Pressure (BP) of 80/50 mm Hg, and SpO2 of 93% on room air. Thirty minutes before giving the skin incision, ceftriaxone 50 mg/kg was administered intravenously.[5]

General anaesthesia was started by preoxygenating the patient for three minutes. Midazolam 1mg, Ondansetron 4mg was given as premedication.

Induction of anaesthesia was performed with 100 μg fentanyl and 6 mg etomidate. 50 μg phenylephrine bolus was used to maintain blood pressure. Endotracheal intubation was performed after achieving muscle relaxation with Vecuronium 4 mg. The patient was intubated with a 5.5-cuffed endotracheal tube. B/L air entry equal. The patient was placed on volume control mode with a tidal volume of 300ml, RR of 10 breaths/min, without Positive End-Expiratory Pressure (PEEP).

Invasive temperature monitoring was done throughout the procedure.

Post intubation, vitals are stable with SpO2 of 100%. Sevoflurane, oxygen, air and Vecuronium top-ups were used to maintain anaesthesia.

An ultrasound-guided 5.5 Fr triple lumen central venous catheter was placed in the right internal jugular vein. Additionally, a 22G peripheral catheter was inserted in the right radial artery for invasive blood pressure monitoring.

During surgery, arterial blood gas analysis revealed a pH of 7.44, a PaO_2 of 107 mmHg, a PCO_2 of 36 mmHg, a base excess of 0.6 mmol/L, a haematocrit of 40%, and electrolytes were within normal limits.

At the end of the procedure, all anaesthetic agents were discontinued. Intravenous paracetamol (15 mg/kg) was administered for pain relief, and neostigmine along with Glycopyrrolate were given to reverse the neuromuscular blockade. Once muscle strength and reflexes had sufficiently returned, the patient was extubated.

The surgery lasted four hours. Fluid management was carried out using crystalloids based on the Holliday-Segar formula. Urine output was 1.5-2 mL/hour, and there was a blood loss of 160 ml. Normothermia was maintained throughout the procedure.

After stable vital signs were achieved, the child was moved to the neuro Intensive Care Unit. Postoperatively, the patient remained stable with no episodes of SVT. Intravenous paracetamol was administered for pain relief, and antibiotics and antiepileptic were started as needed. On postoperative day 10, following the paediatric cardiologist's or consultant's assessment, the child was transferred to the cardiac care unit.

4. Discussion

Patients with cyanotic CHD are prone to develop cerebral abscesses. Survival to the adulthood has increased to 85% with the advancement in surgical and medical management.[6,7] The anaesthetic management includes avoiding myocardial depression while maintaining hemodynamics and vigilant monitoring to prevent air embolism and other complications such as thrombosis, hemorrhage, infective endocarditis, and paradoxical embolism. The ventricular interdependence is lost in univentricular hearts, resulting in abnormal systolic and diastolic function of a single ventricle and is more likely to fail under the stress of surgery and anaesthesia, for which the myocardial depressant drugs should be avoided. We used opioids, etomidate, and sevoflurane, which are considered safe for cardiac patients. Fentanyl was selected for its minimal impact on hemodynamic and pulmonary vasculature, while providing effective analgesia. It is important to avoid prolonged preoperative fasting and ensure adequate hydration to prevent polycythaemia. Additionally, intravenous cannulas and all lines should be properly flushed to prevent air bubbles, as these patients are at higher risk for paradoxical air embolism due to bi-directional shunts. These patients are at increased risk of infective endocarditis and antibiotic prophylaxis was provided.[8] These patients have a higher risk of desaturation because of related pulmonary stenosis. The strategies used for this patient included maintaining a moderately elevated tidal volume (10-15 mL/kg), a low respiratory rate (10 breaths per minute), reducing inspiratory time, and avoiding an increase in positive end-expiratory pressure. Nitrous oxide was avoided to prevent a drop in saturation, and ensuring adequate muscle relaxation helped prevent increased intracranial pressure, cerebral hypoxia, and reduced cerebral blood flow. Intermittent intravenous bolus injections of 50 μg of phenylephrine were used to maintain vascular resistance, blood pressure, and systemic oxygen saturation. The ratio of PVR to SVR influences SpO2. A decrease in PVR increases pulmonary blood flow to the peripheral alveoli, resulting in higher arterial saturation.

Anaesthesiologists needed to manage PVR and SVR using pharmacological and ventilator techniques.[8,9]

5. Conclusion

In patients with congenital cyanotic heart disease (cCHD), the anaesthetic objectives are to maintain systemic vascular resistance (SVR) and avoid elevations in pulmonary vascular resistance to prevent cyanotic spells. Additionally, anaesthesiologists should prevent increases in intracranial pressure (ICP) and ensure that cerebral perfusion pressure is sustained.[10,11] This case shows that with a thorough understanding of pathophysiology and careful planning to prevent potential complications, patients with complex congenital heart disease can be successfully anesthetized for non-cardiac surgery.

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None.

7. Conflict of Interest

None.

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