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CASE REPORT

Hypoxic Spell After Abdominal Surgery in Tetralogy of Fallot Patient: From Being Asymptomatic to Symptomatic

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ABSTRACT

Tetralogy of Fallot is a cyanotic congenital heart disease involving hypoxic episodes. We herein present a case of a patient who underwent the Duhamel operation for Hirschsprung's disease. The patient had hypoxic seizure triggered by surgical stimulation, anesthesia effect, and postoperative agitation and was diagnosed with Tetralogy of Fallot via echocardiography postoperatively.

Keywords: Anesthesia, child, congenital heart diseases, noncardiac surgery, tetralogy of fallot

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Introduction

Hirschsprung's disease refers to the formation of an aganglionic segment in the distal bowel during intestinal development. The surgical treatment option for this disease is the Duhamel operation. Cyanotic heart disease is mainly caused by Tetralogy of Fallot (TOF).[1] TOF has four components: ventricular septal defect (VSD), right ventricular outflow obstruction, overriding of the aortic root over the ventricular septum, and right ventricular hypertrophy. We herein present a case of a patient who was asymptomatic in the preoperative period but was diagnosed with TOF and had hypoxia postoperatively.

Case Report

A 9-month-old boy weighing 9.5 kg was evaluated for a planned Duhamel operation. The patient had no known comorbidities, medication use, dyspnea, hypoxia, cyanosis, spell attack, or diagnosis of TOF. Preoperative echocardiography (ECHO), which was requested for secure anesthesia management, revealed subaortic and wide VSD, 2–2.5-mm-thin patent ductus arteriosus (PDA), significant valvular–supravalvular pulmonary stenosis, and 50% overriding of the aorta on the interventricular septum. Pediatric cardiology consultation suggested preoperative infective endocarditis prophylaxis. In the intraoperative preinduction monitoring, oxygen saturation was found to be 94%; systemic blood pressure, 95/55 mmHg; and pulse rate, 120/min. Mask induction was performed using 8% sevoflurane, followed by intravenous administration of 0.5 µg/kg fentanyl and 0.6-mg/kg rocuronium bromide. The patient was intubated with a 3.5 mm cuffed endotracheal tube. Administration of sevoflurane (1.5%–2%) and remifentanil as analgesic (0.0125–0.1 mcg/kg/min) was initiated for the maintenance of anesthesia. When the mean arterial pressure decreased due to surgical bleeding, intermittent ketamine analgesia was preferred over remifentanil. During the operation, which lasted 4.5 h, fluid balance and maximum allowable bleeding amount were calculated and compensated for. No hypotensive episode occurred. Postoperatively, the patient created a spontaneous sufficient tidal volume and was extubated, and the oxygen saturation was 94%–96%. He was conscious and had spontaneous eye opening, and his motor movement had reached a sufficient power. The patient's spontaneous breathing continued, and when he started crying, saturation began decreasing. Oxygen support was provided, and despite providing positive pressure support, saturation dropped to 45%. The patient's hemodynamics

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was stable, and all possibilities were simultaneously evaluated to determine the differential diagnosis of hypoxia in an asymptomatic patient with a medical history. No lung sound abnormalities, congestion, bronchospasm, or laryngospasm was noted. To rule out pain-related shallow breathing, fentanyl was administered. It was observed that saturation increased when the patient calmed down but dropped again when he started crying. Cardiology consultation was requested in the operating room. ECHO revealed an ejection fraction of 70, VSD of 12.5 mm in the subaortic region with a bidirectional shunt, stenosis due to the muscle band 10-mm below the pulmonary annulus, and 2–2.5-mm-thin PDA; the ECHO report was interpreted as pink TOF. With the recommendation of the cardiology consultant, 20-mL/kg bolus fluid replacement and 1-mg/kg sodium bicarbonate were administered to the patient. Initiation of postoperative propranolol was suggested. The patient was reintubated and transferred to the intensive care unit. After being sedated and receiving propranolol in the intensive care unit, the patient was extubated on postoperative day 4. After consultation with the cardiovascular surgeon, following angiography, elective TOF correction surgery was scheduled.

Discussion

The present case demonstrates how an asymptomatic TOF patient can become symptomatic after major surgery and anesthesia-related hemodynamic changes.

TOF is characterized by hypercyanotic episodes (hypoxic spells). Some studies have reported cases that were not diagnosed with TOF until adulthood despite prenatal screening.[1] Anesthesia management of these patients is challenging and requires understanding of the pathophysiology of the disease. To reduce agitation, premedication is preferable. However, it can trigger hypoxic spells in patients with TOF due to secondary hypoxia, hypercarbia, and increased pulmonary vascular resistance (PVR). In patients with shunt lesions, care should be taken to ensure there are no air bubbles in intravenous fluid therapy as they can enter the arterial system and cause paradoxical air embolism.[2] Since pulmonary perfusion is low in patients with right-to-left shunts, inhalation induction is slow and intravenous induction is rapid.[3] In these patients, neuroaxial blocks should be avoided to prevent a decrease in systemic vascular resistance (SVR). Furthermore, in this patient population, the goal is to reduce PVR and increase pulmonary artery blood flow and lung perfusion to prevent hypoxia. Because a decrease in SVR or an increase in PVR in bidirectional shunts will increase the right-to-left shunt transition, hypoxia may occur. In the present case, the hypoxic spell may have been caused by exposure to an anesthetic agent that decreases SVR and increases endothoracic pressure with mechanical ventilation, which decreased venous return to the right ventricle. Surgical factors such as prolonged surgical duration, bleeding, and decreases in mean arterial pressure and intravenous volume can also play a role. Agitation, crying, and stress after extubation can make an asymptomatic TOF patient visibly cyanotic. To reduce the PVR/SVR ratio during surgical procedures in patients with TOF, the occurrence of hypercarbia, acidosis, and hypoxia should be avoided.[4] In addition, hypotension should be prevented, and anesthetic agents to be used should be carefully selected. Ketamine is often preferred owing to its ability to increase SVR. The effects of anesthetic agents on PVR and SVR are presented in Table 1.

Table 1. Effect of anesthetic agents on PVR and SVR[5]

Anesthetic drug	SVR	PVR	Dose
Potent volatile agent	↓	↓	0.5–1 MAC
Nitrous oxide	–	↑	
Opioids			
Fentanyl	↓		1–2 mcg/kg
			0.5–2 mcg/kg/hr
Morphine	↓		0.05–0.2 mg/kg
			0.02–0.2 mg/kg/hr
Midazolam	–	–	0.1 mg/kg (IV)
Ketamine	↑	–	1–2 mg/kg (IV)
			5–20 mcg/kg/min
Propofol	↓	–	2–3 mg/kg
			100–300 mcg/kg/min
Dexmedetomidine	↑–↓	↓	0.2–1 mcg/kg/hr

↑: Increase; ↓: Decrease; PVR: Pulmonary vascular resistance; SVR: Systemic vascular resistance; MAC: Minimum alveolar concentration; IV: Intravenous

Intravenous fluid bolus should be administered to improve right ventricular filling and pulmonary flow. Furthermore, beta-blockers should be used to help reduce infundibular spasm, improve right ventricular outflow obstruction, and increase pulmonary flow. Conversely, phenylephrine should be administered to increase systemic afterload, with the patients placed in the knee–elbow position. Mild compression of the intraoperative abdominal aorta has the same effect as the knee–elbow position. To prevent postoperative agitation, morphine can be used.[1] In the present case, intravenous volume was provided through fluid bolus and erythrocyte suspension replacement. Ketamine was used to increase SVR, fentanyl to prevent agitation under operating room conditions, and sodium bicarbonate to address acidosis. Subsequently, sedation was deepened, and the patient was reintubated and transferred to the intensive care unit with mechanical ventilator support for better control of hypercarbia and hypoxia.

Conclusion

In the anesthesia management of patients with TOF, intravenous volume and SVR must be preserved, conditions and medications that increase PVR must be avoided, and hemodynamic stability must be ensured. Ketamine is a good treatment of choice as it increases SVR. Joint assessment by an anesthesiologist, cardiologist, pediatrician, and cardiovascular surgeon is recommended when planning surgical procedures for these patients. Perioperative management of patients with TOF relies on a thorough understanding of the pathophysiology of the disease, planned operation, and effects of the anesthetic drugs used.

Disclosures

Informed Consent: Written informed consent was obtained from patient's family.

Peer-review: Externally peer-reviewed.

Conflict of Interest: None declared.

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