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TYMPANOPLASTY IN ADOLESCENCE WITH UNREPAIRED TETRALOGY OF FALLOT (TOF): ANAESTHETIC MANAGEMENT

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ABSTRACT

TOF is the commonest cause of cyanotic congenital heart diseases (CHD) in children. A 15-year old male patient with unrepaired tetrology of fallot was scheduled for tympanoplasty. Patient had pulmonary stenosis and bulging anterior chest wall on inspection. Patient was administered for standard general anaesthesia without nitrous oxide. Patient was extubated following an uneventful procedure. The challenge for anesthesiologists in handling patient with CHD for non cardiac surgery relies on the patient's age, complexity of the heart lesion coupled with patient's capacity to compensate, urgency of surgery and multiple coexisting diseases. Understanding anaesthetic management and pathophysiological intertactions are keys to successful management of these patients.

Key words: Adolescence, Tetrology of fallot, Tympanoplasty.

INTRODUCTION

Congenital heart disease (CHD) occurs in 0.5-1 % of all live births [1]. TOF is the commonest cause of cyanotic CHD in children accounting for 10% of CHD. Nonoperated TOF patients have a survival rate of 30% at 10 years and < 3% at 40 years [2]. The condition is composed of four parts: membranous ventricular septal defect (VSD), right ventricle outflow tract (RVOT) stenosis, right ventricular hypertrophy (RVH), overriding of aorta [3].

The long term effects of congenital heart diseases continue as the child gets older, either before repair or in the unusual case of a patient who has not undergone repair. Compensatory polycythemia increases with time and results in an increased incidence of pulmonary, renal and thrombotic events. Cardiomyopathy arising from right ventricular hypertrophy and failure is the usual cause of death in adulthood [4, 5].

Blood flow is preferentially shunted to the heart, brain, and kidney with decreased flow to skin, muscle, bone, and splanchnic circulation, resulting in decreased somatic growth and increased metabolic rate [6].

The most dangerous consequences of this patho-physiology is a hypercyanotic episode or "tet" spell, that results from a sudden decrease in systemic vascular resistance (SVR) or a sudden increase in oxygen consumption [6].

CASE HISTORY

Preoperative period

A 15-year old, 47 kg adolescence was admitted in hospital for tympanoplasty complaining of pus discharge from left ear since 3-4 years, associated with episodes of chest pain since 3-4 months. He had a history of unrepaired tetralogy of fallot with significant pulmonary stenosis. The physical examination revealed a grade II pansystolic murmur at left sternal border in the 2nd–3rd intercostal space on auscultation with bulging anterior chest wall on inspection.

An electrocardiogram (ECG) showed suspected left ventricular hypertrophy (LVH) and RVH with minor right axis deviation with large t wave in V_3, V_4, and V5. A 2D-Echo finding suggested a large subaortic VSD with 50% aortic override with bidirectional shunt, severe valvular and infundibular pulmonary stenosis, and left

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aortic arch, moderate tricuspid and mitral regurgitation were seen. RVOT Gmax: 87mmhg, LPA: 15mm, RPA: 14mm, DTA: 13mm. The chest X-ray showed mildly increase pulmonary vascularity with rib notching.

The X-ray bilateral mastoid suggested left mastoiditis with cholesteatoma. The patient had normal range of serum electrolyte, renal function test and liver function test. The patient's arterial blood gas (ABG) analysis results revealed PH-7.451, PaO_2 -74 mmHg, $PaCO_2$-38 mmHg, HCO_3 -23.9, Base excess-1.7 mmol/l and O_2 saturation- 89%. The cardiologist recommended infective endocarditis prophylaxis and maintenance of SVR in the perioperative period.

Perioperative period

The patient was prepared for surgery with standard peripheral line access. Baseline hemodynamic studies were heart rate-78/min, blood pressure-110/62 mmHg, on air O_2 saturation- 90%.

Patient was premedicated with injection glycopyrrolate 0.2 mg, ondansetrone 4 mg, and fentanyl 50 µg intravenously. Anaesthesia was induced with injection ketamine 100 mg intravenously and after confirming mask ventilation, injection succinylcholine 100 mg was administered, and the airway was secured with 7.5 mm oral endotracheal tube. After intubation vitals were heart rate-130/min, blood pressure- 146/98 mmHg and O_2 saturation-100%. Anaesthesia was maintained with oxygen, sevoflurane and titrated doses of vecuronium; nitrous oxide was avoided. The intraperative course was uneventful with minimal blood loss and lasted for 2 hr. IV fluids administered were normal saline and Ringer's lactate, one liter each. Recovery was unremarkable, and trachea was extubated after reversing the residual neuromuscular blockade and after meeting the criteria for extubation.

Postoperative period

Patient was shifted to intensive care unit for continuous monitoring. For postoperative analgesia injection diclofenac 75 mg intramascular was administered. Postoperative ABG revealed PH- 7.357, PaO2- 96.7, $PaCO_2$- 44.7, HCO_3- 24.3, Base excess-1.1 and O_2 saturation- 99. No anaesthetic and surgical complication occurred. Patient was called for follow up examination after one week and there were no any problems with anaesthesia or surgery during this period.

DISCUSSION

Anaesthesia for the adolescence with tetralogy of fallot requires careful preparation of the operating room. Cardiac drugs should be immediately available and the anaesthesia machine must be checked carefully preoperatively because of the low margined for error in these patients. Adequate oxygenation is essential to the survival and well-being of this patient in surgical setting [7]. Infective endocarditis is a major concern for all patients with congenital heart disease. Sterile technique in line insertion and prophylaxis infective endocarditis with antibiotics is indicated.

The goal of anesthetic management in patients with TOF is aimed towards: (i) maintenance of normovolaemia (ii) avoidance of a decrease in SVR and (iii) avoidance of an increase in PVR. The "tet" spells or hypercyanotic attacks are generally triggered by decrease in SVR or a spasm of cardiac muscle in the region of the RVOT, resulting in an increase in the magnitude of the R→L shunt. Traditional treatments are alpha agonists and fluids to increase SVR and beta-adrenergic blocking agents to decrease infundibular spasm.

Monitoring needs for cyanotic patient undergoing noncardiac surgery include ECG, noninvasive blood pressure monitoring (NIBP), pulse oximetry, end tidal capnography ($EtCO_2$), temperature, and airway pressure. Additional invasive monitoring including direct arterial cannulation, central venous pressure, trans-esophageal echocardiography and urinary catheterization may be warranted if the patient's cardiovascular status or the planned surgery may result in hemodynamic instability [7]. In presence of right to left shunt the end-tidal carbon dioxide reading consistently underestimate $PaCO_2$, and this discrepancy also worsens with hypoxemia. The lower end-tidal carbon dioxide is explained by relatively large dead-space ventilation caused by the right to left shunt [8].

The choice of the anaesthetic agents are governed by their hemodynamic consequences. The rate and dose are more important than the actual drug used. Sedative premedication is helpful to reduce myocardial oxygen demand, but care must be taken in cyanotic as the SpO_2 level may drop below the acceptable limits. The most cardio stable induction agent is etomidate [10]. Its beneficial effects include rapid onset of action, rapid recovery and maintenance of cardiovascular stability in both healthy and hemodynamically compromised patients. Ketamine can also be used as an induction agent owing to its central sympathetic stimulation properties. At lower concentration, the administration of volatile anesthetic improves arterial oxygenation by causing relaxation of the muscle spasm in RVOT, and decreases the total body oxygen consumption [11]. The SVR must be maintained or even increased, while the PVR is decreased to increase pulmonary blood flow and function of right side of the heart. Because most intraoperative manipulations increase PVR (such as sympathetic stimulation and encroachments on lung volumes), ventilatory control is crucial. Since such control is under the disposition of the anaesthetist, it can be manipulated by the use of 100% oxygen and hypocapnia to decrease PVR [7]. To increase the pulmonary blood flow factors that increase PVR should be avoided, i.e. hypoxia, hypercarbia, hypothermia, acidosis, increased sympathetic tone and use of sympathomimetic drugs. The SVR should be aggressively maintained by the use of presser agents, such as phenylephrine, if needed. Increases in SVR decrease right- to- left shunting and improve arterial

oxygen saturation [7]. Maintaining normothermia is mandatory as hypothermia may induce pulmonary vasoconstriction and coagulation disorders. Adequate pain control is essential to avoid pain induced increase in sympathetic tone.

Alterations in pharmacokinetics and pharmacodynamics occur in the patient with tetralogy of fallot, as with any cyanotic heart lesion with right-to-left shunting. Theoretically, the speed of intravenous induction in these patients would be shortened, because systemic venous blood bypasses the pulmonary circuit and, therefore appears in the arterial blood quicker. Inhalational agent uptake would be slower due to the diminished pulmonary blood flow [7].

The use of nitrous oxide as an adjunct to anaesthesia is controversial in these patients. Nitrous oxide is thought to increase PVR in adults but not in children; any intravascular air bubbles will expand in the presence of nitrous oxide and therefore increase the deleterious effect of air emboli [9].

CONFLICT OF INTEREST STATEMENT

No conflict of interest.

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