CASE REPORT

Anesthetic Management for Incomplete Atrioventricular Septal Defect

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ABSTRACT

Background: Atrioventricular septal defect (AVSD) is a congenital heart anomaly that arises from the incomplete fusion of the endocardial cushions, resulting in communication between the atria and ventricles and abnormal development of the atrioventricular valves. Incomplete AVSD accounts for a smaller proportion of congenital heart defects but presents significant challenges in both surgical and anesthetic management due to complex pathophysiology and perioperative risks.

Aim: To report and discuss the anesthetic management of a pediatric patient with incomplete AVSD undergoing surgical repair.

Case: We report the case of a 7-year-old boy with incomplete AVSD who underwent surgical closure and mitral valve cleft repair. Preoperative evaluation included echocardiography and cardiac catheterization. General anesthesia was induced with fentanyl, propofol, and sevoflurane, and maintained during cardiopulmonary bypass (CPB). Intraoperative transesophageal echocardiography (TEE) confirmed effective repair. Postoperatively, the patient experienced transient arrhythmia that resolved with pacing and was successfully extubated on the first postoperative day without complications.

Discussion: Children with congenital heart disease (CHD) are at higher risk of morbidity and mortality due to the complex physiological derangements caused by the defects. Anesthetic management in AVSD depends on the degree of left-to-right (L to R) shunting and the presence and severity of pulmonary vascular hypertension. Important considerations include neonatal and pediatric anesthesia principles, congenital cardiac anatomy and physiology, CPB techniques, and potential postoperative complications

Conclusion: Anesthetic management in incomplete AVSD requires a comprehensive understanding of pediatric CHD, perioperative monitoring, and CPB protocols. Multidisciplinary collaboration and meticulous perioperative planning are crucial in improving outcomes and minimizing complications in pediatric cardiac surgery.

Keywords: atrioventricular septal defect; cardiopulmonary bypass; congenital; congenital heart disease; general anesthesia

INTRODUCTION

Congenital heart disease (CHD) refers to structural or functional abnormalities of the heart present at birth. The incidence of CHD is approximately 8–10 per 1000 live births. Left-to-right (L to R) shunt lesions are the most common congenital heart defects, accounting for approximately 50% of all lesions.[1] All L to R shunts produce a greater volume load on the cardiovascular system, which increases the risk of a suboptimal outcome if not properly treated. Atrioventricular septal defect (AVSD) is a spectrum of cardiac malformations resulting from incomplete fusion of the endocardial cushions. The predicted incidence of AVSD ranged from 0,24 to 0,31 per 1000 live births, which accounts for 4% of congenital heart defects. It could be classified as either a complete or an incomplete AVSD based on the septal defect.[2,3]

CHD refers to structural or functional abnormalities of the heart that are present at birth. The incidence of CHD is approximately 8–10 per 1,000 live births. Left-to-right (L to R) shunt lesions are the most common type of congenital heart defect, accounting for about 50% of all cases.[1] All L to R shunts result in a volume overload on the cardiovascular system, which, if left untreated, may lead to poor outcomes. AVSD is a spectrum of cardiac malformations resulting from the incomplete fusion of the endocardial cushions. The incidence of AVSD is estimated to range from 0.24 to 0.31 per 1,000 live births, accounting for approximately 4% of congenital heart defects. AVSD may be classified as either complete or incomplete, depending on the extent of the septal defect.[2,3]

AVSD repair requires surgical intervention that carries a significant risk of complications, posing challenges to both cardiac surgeons and anesthesiologists. Anesthesia for CHD, including AVSD, necessitates a comprehensive understanding of neonatal and pediatric anesthesia principles, the anatomy and physiology of congenital heart defects, cardiopulmonary bypass (CPB) principles, and the expected postoperative complications following pediatric cardiac surgery.[2] Children with AVSD experience significant morbidity and mortality due to risks such as postoperative left atrioventricular valve regurgitation, residual intracardiac shunting, pulmonary hypertension, and various life-threatening arrhythmias. Improvements in AVSD surgical outcomes have been attributed to advances in perioperative management, including CPB techniques, myocardial protection, anesthesia, and postoperative care.[4] Hence, this report highlights our experience with the anesthetic considerations in managing a patient with incomplete AVSD undergoing surgical repair.

CASE

A 7-year-old boy with an incomplete AVSD was admitted and scheduled for surgical repair. He had no prior history of cyanotic spells, suboptimal weight gain, or developmental delay. The AVSD was diagnosed when the patient complained of cough, shortness of breath, and unresolved fever one year before admission. Echocardiography detected an incomplete AVSD, a large atrial septal defect (ASD) L to R, and a large inlet to outlet ventricular septal defect (VSD) L to R.

He underwent cardiac catheterization, which confirmed the diagnosis (Figure 1), which was then controlled conservatively with active follow-ups. Two months before admission, follow-up echocardiography showed an incomplete AVSD with large primum ASD, small inlet to outlet VSD, moderate TR, severe MR, and mild PR. Hence, the patient was advised and has agreed to surgical ASD and VSD closure with mitral cleft repair.

A complete preoperative assessment was carried out. He weighed 15 kg, vital signs were within normal limits, while physical examination showed a positive cardiac murmur. Complete blood count showed no abnormality, and chest x-ray showed an enhanced vascular marking with enlargement of the left atrium and both ventricles (Figure 2). The patient was classified as American Society of Anesthesiologists (ASA) III, and general anesthesia with a central venous catheter (CVC), arterial line, and transesophageal echocardiography (TEE) were programmed for anesthesia. After analgetic sedation, an arterial line was established using a 22-G catheter at the left brachial artery. Induction was commenced with 60 mcg fentanyl, 20 mg propofol, inhaled 1% sevoflurane, and 20 mg rocuronium. A 5.5 Fr endotracheal tube was placed and connected to the ventilator, set with pressure control mode, pins 10 RR 25 PEEP 3 I: E 1:1,5. CVC was placed in the right internal jugular vein.

Preoperative TEE measurement showed an ejection fraction of 60%, a 6 cm ASD primum L to R shunt, a 4 cm VSD inlet L to R shunt, severe MR with a 1,6 mm cleft on the anterior mitral leaflet (AML), and moderate TR with 4 mm VC. Cardiopulmonary bypass (CPB) was initiated while the anesthesia was maintained using 1% sevoflurane. Serial blood glass analysis was collected for monitoring (Table 1). Intraoperative hemodynamic parameters were stable, and the surgical repair was uneventful. CPB was maintained for 125 minutes, and the aortic cross-clamp was used for 98 minutes. Post-repair TEE measurement showed an ejection fraction of 58%, a 4 mm residual VSD, moderate MR with 5 cm VC, and moderate TR with 5 cm VC. Hence, the patient was initiated and weaned from CPB using 5 mcg/kgBW/min dobutamine support, 50 mg protamine, 100 cc of PRC, 100 cc of TC, and 125 cc of FFP. He experienced an arrhythmia after CPB weaning but was successfully treated with pacing. Anesthesia and surgery took a total of 270 minutes, and the patient was transferred to the intensive care unit (ICU) while still sedated.

The patient regained consciousness 20 hours after surgery and could tolerate minimal ventilator support. The recovery was uneventful with stable hemodynamics. He was supported with 3 mcg/kgBW/min dobutamine and was given IVFD D51/2NS 30 cc/hour, ceftriaxone 450 mg/12 hour IV, continuous fentanyl IV, paracetamol 300 mg/8 hour IV, furosemide 7.5 mg/12 hour IV, omeprazole 15 mg/24 jam IV, methylprednisolone 20 mg/24 jam, and fluid balance/2 hour with target 0 to -500 cc/24 hour. The complete blood count was within normal limits. Serial blood gas analysis (BGA) per 8 hours was obtained for monitoring (Table 2). He was extubated on the first postoperative day. The patient was then transferred to the high care unit (HCU) on the second postoperative day.

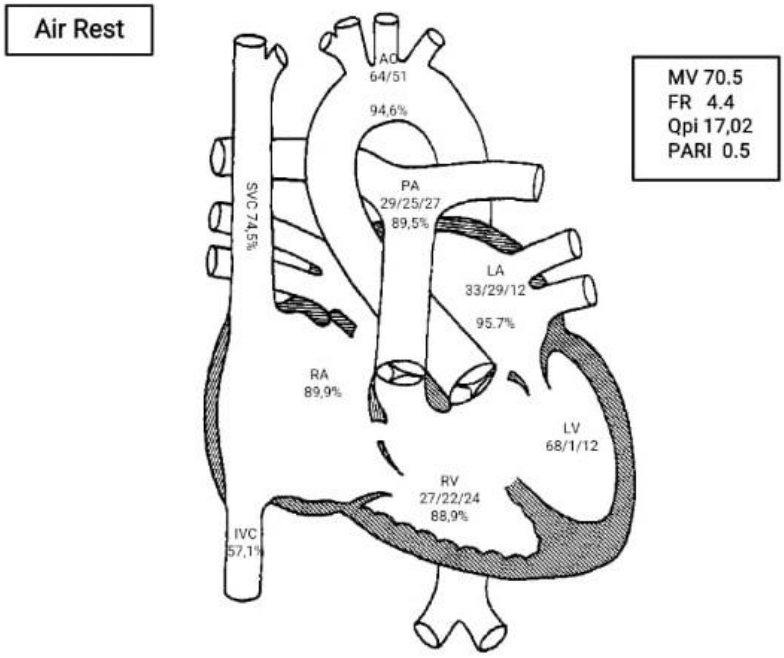

Figure 1. Cardiac catheterization showed an incomplete AVSD

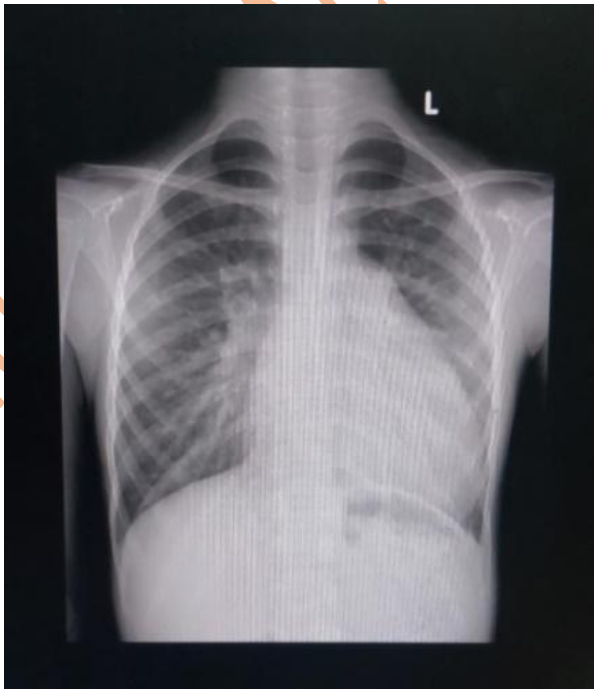

Figure 2. Chest x-ray showed enhanced vascular marking with enlargement of the left atrium and both ventricles

Table 1. Arterial blood gas analysis

Blood gas	Preoperative	Post-CPB
pH	7.375	7.345
PCO2 (mmHg)	33.3	43.0
PO2 (mmHg)	226	80
BEecf (mmol/L)	-6	-2
HCO3 (mmol/L)	19.5	23.4
TCO2 (mmol/L)	21	25
sO2 (%)	100	95
Lac (mmol/L)	1.70	2.33
Na (mmol/L)	140	143
K (mmol/L)	2.8	3.4
Cl (mmol/L)	107	107
iCa (mmol/L)	1.22	1.32
TCO2 (mmol/L)	18	22
Glu (mg/dL)	95	106
BUN (mg/dL)	10	8
Crea (mg/DL)	<0,2	<0.2
Hct (%PCV)	29	32
Hb (g/dL)	9.9	10.9

Table 2. Arterial blood gas analysis

Analysis	ICU 1	ICU 2	ICU 3	Post-Extubation 1	Post-Extubation 2	Post-Extubation 3
FiO2	50%	50%	50%	30%	30%	30%
pH	7.363	7.413	7.437	7.373	7.355	7.384
PCO2	47.3	44.7	32.2	45.7	50.3	50.5
PO2	180	185.1	152.9	155.9	105	109
AaDO	116.8	115.8	161.4	0	48.1	43
BEecf	0.55	2.84	-2.31	0.57	1.47	3.51
HCO3	26.3	27.9	22.5	24.7	27.5	29.5
sO2	99.7	100 %	99.8%	100	99	98.6
Lac	2.2	1.9	2.3	1.9	1.3	1.3
ScVO2	92.1	67.5	69.7	-	-	-

DISCUSSION

Children with CHD are at higher risk of morbidity and mortality compared to their healthy peers, due to the complex physiological derangements caused by the defects. These anomalies pose significant surgical and anesthetic challenges. In this case, we presented the anesthetic management of a 7-year-old child with an incomplete AVSD undergoing ASD closure, VSD closure, and mitral cleft repair. AVSD is a congenital cardiac anomaly resulting in a connection between both atrial and ventricles. This results in a one-sided workload increase that could cause arterial desaturation and chronic fluid overload. Anesthetic management in AVSD depends on the degree of L to R shunting and the presence and severity of pulmonary vascular hypertension. Our patient had a transitional or incomplete AVSD; thus, anesthetic principles similar to those for ASD and AV valve regurgitation were applied. Important considerations include neonatal and

pediatric anesthesia principles, congenital cardiac anatomy and physiology, CPB techniques, and potential postoperative complications.[5–9] Preoperative assessment includes blood work, electrocardiogram (ECG), chest radiography, echocardiography, and angiography, usually performed 1–2 weeks before surgery. Midazolam may be administered for premedication. In our case, these assessments were completed, including cardiac catheterization, and a preoperative visit was performed one day before surgery. Anesthetic induction commonly involves intravenous or inhalational agents. Ketamine is preferred for induction as it preserves systemic vascular resistance (SVR) and mean arterial pressure (MAP). Etomidate and propofol may be used cautiously due to the risk of decreased SVR and MAP. Rocuronium is the preferred muscle relaxant. Maintenance was achieved with Sevoflurane and titrated fentanyl. In our case, the patient was induced with 60 mcg fentanyl, 20 mg propofol, inhaled 1% sevoflurane, and 20 mg rocuronium to facilitate intubation. ASA recommends standard monitoring for cases involving CPB using invasive arterial and central venous lines, TEE, cerebral oximetry, and urine output.[10]

Intraoperative arterial blood gas and ACT were within normal limits, so CPB was initiated. During CPB, ventilation is discontinued; however, anesthesia, analgesia, and muscle relaxation must be maintained. Isoflurane (or sevoflurane) is added to the CPB circuit at a concentration of 0.5–1.0%, or a propofol infusion may be administered. Additional opioids and muscle relaxants are given before the initiation of CPB or incorporated into the main CPB circuit. Further doses of benzodiazepines may also be used. Depending on the type of surgery, surgical technique, and/or surgeon preference, the perfusionist may allow the patient's core temperature to drop to 34–35°C (referred to as 'warm shortcut') or actively cool the patient to 32°C or lower (down to a minimum of 15°C). Perfusion pressure, venous saturation, cerebral oxygenation, hematocrit, and electrolyte and acid-base status are continuously monitored. Target perfusion pressures vary by age and typically range from a MAP of 30 to 50 mm Hg (from neonates to young adults).[11] Postoperative TEE confirmed surgical results, hence separation from CPB followed criteria including normothermia, stable rhythm, ventilatory readiness, and perfusion parameters. In our case, the patient experienced arrhythmia after CPB weaning, but it resolved after pacing. He was supported with dobutamine 5 mcg/kgBW/min, and blood products were given accordingly.

After hemostasis was achieved and the surgery was completed, the patient was transferred to the ICU. During transfer, invasive monitoring was continued, and emergency medications and fluids were kept readily available. A comprehensive handover, including surgical details and echocardiographic findings, was communicated to the ICU medical and nursing staff. Maintaining adequate cardiac output is a crucial component of postoperative care. Since direct measurement of cardiac output is difficult in pediatric patients, surrogate markers such as MAP, venous oxygen saturation, and serum lactate are commonly used. Postoperative cardiac complications may include arrhythmias, such as second- or third-degree atrioventricular block requiring pacemaker support, or tachyarrhythmias, including supraventricular tachycardia or junctional ectopic tachycardia.[2] In our patient, postoperative management

included continuous monitoring with ventilatory support, postoperative electrocardiography, echocardiography, chest radiography, fluid and urine output monitoring, drain assessment, serial arterial blood gas analyses, and routine laboratory testing. The patient remained in the ICU for two days; extubation was performed on postoperative day one, and the patient was transferred to the HCU on the second day. No postoperative complications were noted during the ICU stay.

CONCLUSION

Surgical management of CHD, including AVSD, poses significant challenges for both cardiac surgeons and anesthesiologists. The unique pathophysiological changes associated with AVSD place these patients at higher risk for perioperative morbidity and mortality. Children with AVSD are particularly vulnerable to postoperative complications; hence, the anesthetic management requires a comprehensive understanding of neonatal and pediatric anesthetic principles, the anatomy and physiology of congenital cardiac anomalies, CPB protocols, and common postoperative complications in pediatric cardiac surgery. The anesthetic approach must be tailored based on the degree of L to R shunting and the presence and severity of pulmonary vascular hypertension. Improvements in AVSD surgical outcomes are closely linked to advancements in perioperative management strategies, including refinements in CPB techniques, myocardial protection, anesthesia protocols, and postoperative care. Multidisciplinary collaboration among surgeons, anesthesiologists, perfusionists, and critical care teams plays a vital role in the overall success of AVSD repair.

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