Postoperative complications and their management after arterial switch operation in infants with transposition of great arteries

Li-Xing Zhu, Lin-Hua Tan, Xiao-Jun He, Cai-Yun Zhang, Yan-Ping Yu, Ze-Wei Zhang
Hangzhou, China

300

Background: Arterial switch operation (ASO) has been optimal for children with transposition of great arteries (TGA) in either simple or complex form with an excellent survival rate. This operation was introduced late in China, but there has been a decreasing mortality in recent years. Optimizing the postoperative management has been essential to improve the survival rate after ASO. This study summarizes the experience in the management of the postoperative complications after ASO.

Methods: Twenty-eight infants with TGA underwent ASO from January 2004 through December 2006. These patients aged 1-70 days (median, 6 days) had a body weight of 3.36±0.57 kg on average. Before operation, continuous intravenous infusion of prostaglanding E1 was routinely used to keep the ductus open with a SpO_2 of 75%-90%. Ten patients required tracheal intubation and mechanical ventilation. Two patients underwent emergency ASO under general anesthesia and low-flow cardiopulmonary bypass or hypothermic circulatory arrest. All the 28 patients were further treated with modified ultrafiltration and delayed sternal closure after cessation of cardiopulmonary bypass. After ASO, they were on circulatory and respiratory support, with antibiotics, nutritional supplement in the ICU.

Results: The patients had a cardiopulmonary bypass time of 167±32 minutes and an aortic cross-clamping time of 101±24 minutes. Delayed sternal closure was carried out on 3.63±1.49 days after ASO. They had a mechanical ventilation of 5.89±3.02 days and an ICU stay of 10.12±3.25 days. There were 5 deaths after ASO with a mortality rate of 17.9%. Fifteen patients developed low cardiac output syndrome. In 12 patients presenting with cardiac arrhythmias, 9 had paroxysmal supraventricular tachycardia, 1 had frequent ventricular premature beats, 1 had ventricular tachycardia, and 1 had ventricular fibrillation. One patient suffered from pulmonary hypertensive crisis. Three patients with major bleeding and tamponade required emergency mediastinal exploration and finally survived. There were 6 patients with ventilator-associated pneumonia, 6 with delayed incision healing, and 1 with chylothorax.

Conclusions: The complications after ASO are common and complicated. Understanding of the physiological characteristics of infants and the pathophysiological changes, and optimizing postoperative treatment help to improve the survival rate after ASO.

Introduction

Transposition of great arteries (TGA) is a lethal and relatively frequent malformation accounting for 7%-8% of all congenital cardiac malformations,[1] in which the aorta arises from the right ventricle and the pulmonary artery from the left ventricle. Since Jatene and colleagues performed the first anatomical repair for TGA in 1976, arterial switch operation (ASO) has been optimal in either simple or complex form for children with TGA with an excellent survival rate and good quality of life.[2-5] However, this operation was introduced late in China, with a high morbidity and mortality[6,7] compared to a recent study from Europe.[8] The present study was to evaluate the experience in the management of postoperative complications in 28 infants who had undergone ASO in our surgical intensive care unit (ICU).

Author Affiliations: Surgical ICU, Children's Hospital, Zhejiang University School of Medicine, Hangzhou 310003, China (Zhu LX, Tan LH, He XJ, Zhang CY, Yu YP, Zhang ZW)

Corresponding Author: Lin-Hua Tan, MD, Surgical ICU, Children's Hospital, Zhejiang University School of Medicine, Hangzhou 310003, China (Tel: 86-571-87061007; Fax: 86-571-87033296; Email: linhua_tan@yahoo.com)

301

Methods

Patients

From January 2004 through December 2006, 28 infants with TGA underwent ASO consecutively at Children's Hospital, Zhejiang University School of Medicine, China. The patients, 23 males and 5 females, aged 1-70 days (median, 6 days), had a body weight of 3.36±0.57 kg. Seven (25%) of these infants had ventricular septal defect (VSD), and the remaining 21 (75%) had intact ventricular septum (IVS) (Table).

In ICU, all patients were subjected to cerebral ultrasonography. They received prostaglanding E1 infusion at a low dose of 0.005-0.02 μg/kg per minute to maintain the ductus open, while keeping SpO_2 at 75%-90%. None of them underwent atrial septostomy. Ten patients had tracheal intubation and mechanical ventilation because of cardiopulmonary compromise. Two patients had to undergo emergency ASO because of frequent episodes of cardiac arrest.

Surgical and anesthetic management

A median sternotomy was performed to confirm coronary artery anatomy. Each patient underwent cardiopulmonary bypass or predominantly low-flow hypothermic cardiopulmonary bypass with a limited period of deep hypothermic circulatory arrest if necessary. The great vessels were transected and the internal orifices of the coronary arteries were inspected. The coronary arteries were translocated posteriorly to the neo-aorta. The posterior branches of pulmonary arteries were brought anteriorly to the aorta (Lecompte maneuver) for aortic reconstruction with circumferential suture. At this point, the right atrium was opened and atrial septal defect and/or VSD was repaired. Subsequent reconstruction of the pulmonary artery was performed. The coronary donor sites were "filled in" with a patch of the autologous pericardium. Finally, a continuity between the right ventricle and pulmonary arteries was established with circumferential suture. The patients were treated with modified ultrafiltration and delayed sternal closure[9] after cessation of cardiopulmonary bypass, because capillary leak and edema associated with cardiopulmonary bypass extended into the postoperative period and compromised myocardial and pulmonary function, especially in the immediate postoperative period. The skin was closed over an open sternum, and a plastic (cut chest tube or 3 ml syringe) strut was placed between the sternal halves. Sufficient time was taken to secure hemostasis before closure of the incision. At the same time, we used prothrombin complex at a concentration of 10-20 PE/kg or fibrinogen (0.5 g) and platelet infusion (3 unit-5 unit) after cardiopulmonary bypass.

Leiden coronary artery pattern classification[10] was used to identify the coronary artery anatomy. The classification is based on the origin of the left coronary artery (LCA) and its two branches (AD and Cx) and the right coronary artery (R) from the two facing sinuses of the aorta. The starting point is the non-facing sinus of the aortic orifice. Looking from this sinus towards the pulmonary trunk, the two facing sinuses can be designated counterclockwise as facing sinus 1 (left sinus) and facing sinus 2 (posterior sinus). Both sinuses are separated with a semicolon. For example, the most common coronary type of healthy people is (1AD, Cx; 2R). In this study, 22 infants belonged to (1AD, Cx; 2R), 3 to (1AD; 2R, Cx), 1 to (2R, AD, Cx), 1 to (2AD, Cx; 2R), and 1 to (1R; 2AD, Cx).

Postoperative care

Neuromuscular blockade and continuous narcotic sedation with fentanyl (5-15 μg/kg per hour) continued for at least the first 24 hours after the operation. The anesthetic period was prolonged if there was delayed sternal closure or hemodynamic instability. The patients were monitored by standard surface electrocardiography, pulse oximetry, arterial line, right atrial line and transthoracically placed left atrial and pulmonary arterial catheters. Echocardiography was used to detect residual VSD and left ventricle outlet obstruction, and evaluate left ventricle function, pulmonary hypertension and systemic (tricuspid) atrioventricular valve function.

Inotropics were given as dopamine (5-10 μg/kg per minute), and epinephrine (0.05-0.15 μg/kg per minute) was added when necessary. Milrinone (0.25-0.75 μg/kg per minute) was infused slowly for inotropic support and afterload reduction which was particularly helpful in the immediate postoperative period in these patients.

Sternal closure was done as soon as the patient was clinically stable. Hemodynamic variables including blood pressure, heart rate and arterial blood gas during mechanical ventilation were closely monitored. Additional inotropics were required as necessary during and after the closure. Subsequently, all patients were extubated and transferred out of ICU after having weaned from a ventilator and presenting with stable vital signs.

Table. Patient characteristics

Age	Total cases	TGA/IVS	TGA/VSD
<7 d	14	13	1
8 d-1 mon	10	7	3
1-3 mon	4	1	3

TGA/IVS: transposition of the great arteries with intact ventricular septum; TGA/VSD: transposition of the great arteries with ventricular septal defect.

Results

The patients in this series underwent cardiopulmonary bypass for 167±32 minutes, aortic cross-clamping for 101±24 minutes, and delayed sternal closure on 3.63±1.49 days after ASO. They had a mechanical ventilation time of 5.89±3.02 days and an ICU stay of 10.12±3.25 days.

Five patients (17.9%) died after ASO. Among them, 2 died of severe low cardiac output syndrome (LCOS) secondary to a variant coronary pattern (2R, AD, Cx) and "unprepared" left ventricle respectively, 2 of refractory ventricular arrhythmias, and 1 of pulmonary hypertensive crisis.

The complications of the patients after ASO in our study were different. (1) In 15 patients with LCOS, 3 were extremely severe and complicated by multi-organ dysfunction, and the other 13 (86.7%) were successfully treated. (2) All patients had a transient sinus tachycardia. In 9 patients with paroxysmal supraventricular tachycardia (SVT), 6 were hemodynamically stable, and the other 3 were complicated by hypotension. Sinus rhythm was restored by intravenous bolus of propafenone (0.5-1.5 mg/kg), followed by continuous infusion at a rate of 10 µg/kg per minute. In addition, 3 patients suffered from ventricular arrhythmia: 2 died of sustained ventricular tachycardia and ventricular fibrillation as described above, and 1 who had frequent ventricular premature beats was treated with lidocaine and survived. (3) One patient had pulmonary hypertensive crisis and died. (4) Three patients suffered from persistent bleeding and tamponade during the early postoperative period and required timely mediastinal exploration by opening the sternotomy incision in the ICU. All of them survived. (5) Ventilator-associated pneumonia (VAP) occurred in 6 patients: 2 patients had early-onset VAP (VAP occurred after mechanical ventilation <5 days), with the lower respiratory tract secretions cultured positive in 1 patient with parainfluenza virus III and negative in the other patient; 4 patients had late-onset VAP (VAP occurred after mechanical ventilation ≥5 days), with *Pseudomonas aeruginosa* and influenza virus type A infection in 2 patients respectively and negative in the other 2 patients. Seventeen patients were successfully extubated with mechanical ventilation of 3-14 days (median, 4.5 days). In 6 patients who had re-intubation, 5 were successfully re-extubated with a ventilation of 6-14 days (median, 8 days) and the other patient was successfully weaned from the ventilator for the third time with a mechanical ventilation of 16 days. (6) Delayed superficial wound healing occurred in 6 of the patients with negative culture results. All of these patients were cured with intravenous antibiotics, daily sterilization, and nutritional support. (7) One patient had chylothorax on postoperative day 11, and was cured with 10 mg/kg erythromycin injection into the pleural cavity via a chest tube once every day for 2 days. (8) Neurological complications occurred in one patient who had subependymal germinal matrix hemorrhage preoperatively identified by ultrasound. Postoperatively, the patient had no obvious changes with the degree of subependymal germinal matrix hemorrhage and no abnormal neurological signs.

Discussion

In our study, the mortality rate in infants after ASO was 17.9%, which is higher than that reported by Wetter et al.[11] But there was a decrease in mortality in this study from 33.3% to 14.3% and 12.5% in year 2004, 2005 and 2006 respectively. The reasons may be due to (1) better preoperative care before operation and patient selection for ASO; (2) improvement of surgical skills, and better cardiac protection during cardiopulmonary bypass; (3) optimized postoperative management for preventing and managing complications, such as major bleeding and tamponade, LCOS, pulmonary hypertension and arrhythmias.

Bleeding is a common postoperative complication in patients after ASO. Blood loss in the ICU indicates the need for adequate drainage from the mediastinum to avoid cardiac tamponade while correcting a persistent coagulopathy. In our practice, complete hemostasis, normal coagulation, and avoidance of hypertension were effective in reducing the incidence of hemorrhage. But the use of a draining mediastinal tube failed to prevent this complication. Timely exploration by sternotomy incision in ICU can immediately decompress the pericardial space and restore cardiac output.

LCOS was reported in neonates after ASO, in which the cardiac index fell to less than 2 L/min per m^2 during the first operative night, while pulmonary vascular resistance and systemic vascular resistance rose.[12] The causes for LCOS may be related to arrhythmias, left ventricular dysfunction, myocardial ischemia/reperfusion injury, pulmonary hypertension, and residual cardiac anomaly.

To prevent and treat LCOS, the left ventricle in patients with TGA/IVS should be "prepared" for ASO. Otherwise, fast two-stage ASO by pulmonary artery banding should be used. In TGA/VSD patients, the evaluation of pulmonary hypertension is very important to avoid postoperative pulmonary hypertensive crisis. Coronary artery dysfunction is the most common cause of death after ASO,[13] surgeons should be cautious with the transplantation of the coronary arteries. The use of high-dose milrinone (0.75 µg/kg per minute)

302

Original article

after pediatric congenital heart surgery is reported to reduce the risk of LCOS.[14] Inotrops such as dopamine or lower-dose epinephrine are useful in combination therapy to prevent and treat LCOS. Acute increase in preload may be followed by a significant increase in left atrial pressure and pulmonary edema, and a fall in cardiac output. The strategies treating pulmonary hypertension include reducing stimulation to the sympathetic nervous system, adequate analgesia and sedation, administering supplemental oxygen, treating acidosis, and maintaining mild alkalosis, intravenous vasodilators such as inhaled nitric oxide, milrinone and prostaglanding E1. Treatment of fluid overload with diuretics is not usually initiated until 12 to 24 hours after surgery. Intermittent or continuous infusion of diuretics was commonly used.[15] Once acute renal failure and oliguria or anuria occurs, peritoneal dialysis must be carried out as soon as possible to achieve a net negative fluid balance and electrolyte balance.[16]

To maintain a normal sinus rhythm is of great benefit, because losing atrial-ventricular sequential pacing can lead to a 15% to 20% decrease in cardiac output. In our study, SVT appeared to be the commonest cardiac arrhythmias, and intravenous propafenone was effectively and safely used in patients with SVT and unstable hemodynamics. Ventricular arrhythmias may be a sign of coronary insufficiency, and should be dealt with immediately.

All patients in this study received delayed sternal closure. Age, low birth weight, prolonged mechanical ventilation, severe underlying diseases and poor nutritional status may place the neonates and infants after ASO at a high risk for infection. With standard protocols, none of the patients in this series had mediastinal and sternal wound infection. Once there was evidence of infection, broad-spectrum antibiotics were used according to the antibiotic resistance pattern of the pathogenic microbial isolates cultured from lower respiratory aspirations or wound. Fiberoptic bronchoscopy was helpful in the diagnosis and treatment of patients with severe atelectasis. The neonates and infants were poorly tolerable to increasing work of breath. For patients presenting with persistent short of breath, respiratory secretions, and signs of decompensation of respiratory or heart failure, prompt re-intubation and mechanical ventilation were suggested.

Parental nutrition or eternal nutrition is most important to meet high-energy demand, to maintain normal intestinal microflora, to promote wound healing and to help weaning from the ventilator successfully. Malnutrition is common in neonates and infants after ASO, especially in those who have severe underlying diseases and poor nutritional status preoperatively. In the early period after ASO, they often need restricted fluid intake, but relatively high requirement of inotropes and blood products for bleeding may even worsen the malnutrition. High-calorie additives are widely used. In patients with chylothorax, nutritional support should be highlighted because of the additional need for supplements.

Hypoxic ischemic encephalopathy and intracranial hemorrhage are of great concern in neonates with TGA after ASO. Preoperative hypoxemia and cardio-pulmonary compromise, abnormal coagulopathy, cardiopulmonary bypass, postoperative LCOS and decreased oxygen delivery may place infants at a high risk of neurological complications. Routinely we use vitamin K1 and follow up head ultrasound B and CT scan before and after ASO. In this study, one patient who had subependymal germinal matrix hemorrhage preoperatively did not show any evidence of neurological complication postoperatively. Long-term neurodevelopmental outcome is needed to be followed up.

In summary, the complications after ASO are common and complicated. Optimizing the preventive and treatment strategies helps improve the survival rate of patients after ASO.

References

1 Wernovsky G, Mayer JE, Jonas RA, Hanley FL, Blackstone EH, Kirklin JW, et al. Factors influence early and late outcome of the arterial switch operation for transposition of the great arteries. J Thorac Cardiovasc Surg 1995;109:289-301.
2 Jatene AD, Fontes VF, Souza LC, Paulista PP, Neto CA, Sousa JE. Anatomic correction of transposition of the great arteries. J Thorac Cardiovasc Surg 1982;83:20-26.
3 Hutter PA, Kreb DL, Mantel SF, Hitchcock JF, Meijboom EJ, Bennink GB. Twenty-five years' experience with the arterial switch operation. J Thorac Cardiovasc Surg 2002;124:790-797.
4 Norwood WI, Dobell AR, Freed MD, Kirklin JW, Blackstone EH. Intermediate results of the arterial switch repair. A 20-institution study. J Thorac Cardiovasc Surg 1988;96:854-863.
5 Kirklin JW, Blackstone EH, Tchervenkov CI, Castaneda AR. Clinical outcomes after the arterial switch operation for transposition. Patient, support, procedural, and institutional risk factors. Congenital Heart Surgeons Society. Circulation 1992;86:1501-1515.

303

6   Xu ZW, Liu JF, Zhang HB, Zheng JH, Yan Q, Qiu LS, et al. Analysis surgical results of 113 patients with complete transposition of the great arteries. Zhonghua WaiKe Za Zhi 2007;45:801-804.

7   Liu YL, Hu SS, Shen XD, Li SJ, Wang X, Yan J, et al. Indications of arterial switch operation for complex congenital heart defect with severe pulmonary hypertension and ventriculoarterial discordant connection. Zhonghua Yi Xue Za Zhi 2006;86:23-25.

8   Sarris GE, Chatzis AC, Giannopoulos NM, Kirvassilis G, Berggren H, Hazekamp M, et al. The arterial switch operation in Europe for transposition of the great arteries: a multi-institutional study from the European Congenital heart Surgeons Association. J Thorac Cardiovasc Surg 2006;132:633-639.

9   Tabbutt S, Duncan BW, McLaughlin D, Wessel DL, Jonas RA, Laussen PC. Delayed sternal closure after cardiac operations in a pediatric population. J Thorac Cardiovasc Surg 1997;113:886-93.

10 Massoudy P, Baltalarli A, de Leval MR, Cook A, Neudorf U, Derrick G, et al. Anatomic variability in coronary arterial distribution with regard to the arterial switch procedure. Circulation 2002;106:1980-1984.

11 Wetter J, Belli E, Sinzobahamvya N, Blaschzok HC, Brecher AM, Urban AE. Transposition of the great arteries associated with ventricular septal defect: surgical results and long-term outcome. Eur J Cardiothorac Surg 2001;20:816-823.

12 Hoffman TM, Wernovsky G, Atz AM, Kulik TJ, Nelson DP, Chang AC, et al. Efficacy and safety of milrinone in preventing low cardiac output syndrome in infants and children after corrective surgery for congenital heart disease. Circulation 2003;107:996-1002.

13 Yacoub MH. The case for anatomic correction of transposition of the great arteries. J Thorac Cardiovasc Surg 1979;78:3-6.

14 Hoffman TM, Wernovsky G, Atz AM, Bailey JM, Akbary A, Kocsis JF, et al. Prophylactic intravenous use of milrinone after cardiac operation in pediatrics (PRIMACORP) study. Prophylactic Intravenous Use of Milrinone After Cardiac Operation in Pediatrics. Am Heart J 2002;143:15-21.

15 Van der Vorst MM, Ruys-Dudok van Heel I, Kist-vaan Holthe JE, den Hartigh J, Schoemaker RC, Cohen AF, et al. Continuous intravenous furosemide in haemodynamically unstable children after cardiac surgery. Intensive Care Med 2001;27:711-715.

16 Sorof JM, Stromberg D, Brewer ED, Feltes TF, Fraser CD Jr. Early initiation of peritoneal dialysis after surgical repair of congenital heart disease. Pediatr Nephrol 1999;13:641-645.

Original article

304