Common arterial trunk: current implementation of the primary and staged repair strategies

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Original Article

Abstract

OBJECTIVES: In this study, we report our experience on the primary and staged surgical approaches for common arterial trunk (CAT) repair.

METHODS: Between August 2003 and February 2015, 16 consecutive patients underwent CAT repair in our institution. Two different approaches have been followed: group ‘primary repair’ (PR) consists of patients suitable for straightforward CAT repair, who underwent surgery electively at 1–3 months of age (n = 13); group ‘staged repair’ (SR) consists of critically ill neonates with CAT and poor preoperative status or coexisting interrupted aortic arch (n = 3). They underwent staged CAT repair with aortic arch repair and right ventricular-to-pulmonary artery (RV-PA) shunt within the neonatal period, followed by an intracardiac repair later in infancy.

RESULTS: Median age at initial surgical treatment was 8 days (range: 7–21 days) in group SR and 34 days (range: 14–91 days) in group PR (P = 0.03). Mean Aristotle Comprehensive Complexity score was 11 ± 0.6 (range: 11–13) in group PR and 18 ± 3.1 (range: 15–21) in group SR (P < 0.01). Follow-up was completed with a median duration of 3.6 years (range: 8 months to 11 years). There was neither early nor late mortality in both groups. In group SR, the median interval to second stage surgery was 216 days (range: 216–260 days). Seven patients (54%) in group PR required reoperation for RV-PA conduit failure (n = 4), truncal valve repair/replacement (n = 2) or both (n = 1). After initial surgery, Kaplan–Meier freedom from reoperation after 1, 2 and 8 years was 77 ± 12, 68 ± 13 and 20 ± 17% in group PR, and 0% in group SR (log-rank P < 0.01). Although all patients in group SR required reoperation to complete the anatomical correction (second stage procedure), there was no surgical reintervention of truncal valve and aortic arch thereafter.

CONCLUSIONS: Routine elective CAT repair could be safely performed at 1–3 months of age. However, neonatal CAT repair could be associated with a higher mortality especially in the presence of an interrupted aortic arch. In such cases, a staged CAT repair seems to be associated with favourable postoperative course and improved hospital survival, despite the inevitable need for reoperation, which can be performed at a relatively low risk.

Keywords: Congenital heart disease • Truncus arteriosus • Interrupted aortic arch

INTRODUCTION

Surgical management of common arterial trunk (CAT) has evolved considerably. Nevertheless, controversies remain concerning the timing of surgery and the optimal management of associated lesions [1, 2]. Since 2008, we have aimed to perform complete repair at 1–3 months of age for patients with straightforward CAT. However, patients with moderate-to-severe truncal valve regurgitation, interrupted or hypoplastic aortic arch, poor preoperative haemodynamic status and multiple organ failure typically require early repair in neonatal period that is associated with higher risk [3, 4]. In such cases, staged repair (SR) strategies might be helpful to reduce perioperative mortality and morbidity. Staged CAT repair at our institution includes an initial aortic arch repair (AAR) and right ventricular-to-pulmonary artery (RV-PA) valveless conduit, followed by an intracardiac repair later in infancy (Fig. 1A–C). In this study, we evaluate the feasibility and outcomes of both strategies in CAT repair within a single institution.

METHODS

Study design

After Institutional Review Board approval and a waiver of individual consent, a retrospective review of medical records was performed to identify children who had undergone CAT surgery. Surgery was performed between August 2003 and February 2015 in our institution. This limited study period was chosen to reduce era-related changes in surgical and perioperative
management. Only patients with CAT were analysed in this series; patients presenting with hemitruncus, in which one PA branch arises from the aorta whereas the main PA with the other PA branch arises from the RV, were excluded. Although complete primary repair (PR) at 1–3 months postnatally represents our preferred strategy in straightforward CAT, a staged CAT repair was chosen in complex cases with poor preoperative status or coexisting interrupted aortic arch (IAA) that require surgery during neonatal period (within the first 28 days postnatally). Thus, two groups were formed according to the chosen treatment strategy: group PR and group SR.

Surgical technique

All cases were approached via median sternotomy, utilizing cardiopulmonary bypass (CPB). Both PA branches were snared at the onset of bypass, and myocardial protection was ensured by Bretschneider’s solution (Custodiol®, Dr Franz Köhler Chemie GmbH, Bensheim, Germany). Care was taken to excise the PA from the truncal vessel without injuring the coronary arteries. The resultant defect was closed with a bovine pericardial patch. The ventricular septal defect (VSD) was closed with a patch (bovine pericardium or Dacron) and the right ventricular outflow tract (RVOT) was reconstructed with either direct RV-PA anastomosis or a valveless/valved RV-PA conduit. The method of RVOT reconstruction was chosen according to patients’ anatomy and availability of conduits.

A two-stage repair has been used in critically ill neonates with CAT and coexisting IAA (Fig. 1A), using a limited period of moderate hypothermia (25°C) and unilateral antegrade cerebral perfusion. The arterial cannula was inserted into a 3.5 mm GoreTex shunt (W. L. Gore & Associates, Flagstaff, AZ, USA), which was anastomosed end-to-side to the innominate artery. After complete resection of the ductal tissue, an end-to-end anastomosis of the distal aortic arch and posterior wall of the proximal descending aorta was performed. This was followed by anterior patch augmentation, using a large bovine pericardial patch. AAR was followed by implantation of a 6.0 mm valveless GoreTex conduit (W. L. Gore & Associates) as an RV-PA connection (Fig. 1B). VSD closure as well as placement of a larger RV-PA valved conduit was performed later in infancy (Fig. 1C).

Postoperative care

Management of pulmonary vascular resistance is of utmost importance for this difficult subset of patients in order to prevent pulmonary hypertensive episodes and crises. Patients were kept intubated, moderately hyperventilated, with a target systemic arterial pH of 7.50–7.55. They were also maintained on sedation with fentanyl for the first 24–48 h, or until extubation. Inhaled nitric oxide and iloprost were available for the treatment of pulmonary hypertensive crises.

Follow-up

Follow-up was accomplished by routine check-up in outpatient clinic or by direct contact with the referring paediatric cardiologist. ‘Early’ was considered for events that occurred within 30 days after surgery or during the same hospital admission. Follow-up was completed up to February 2015; key aspects during follow-up included postoperative development of truncal valve stenosis or regurgitation, recurrent aortic stenosis as well as reoperation rates in general.

Statistics

Patients’ data were analysed in IBM® SPSS® Statistics Version 21 (IBM Corporation, Armonk, NY, USA). Analysis of time-related freedom from reoperation was performed using the Kaplan–Meier method. A P-value less than 0.05 indicated a statistically significant difference.

RESULTS

Patient demographics, morphological and procedural data and outcome are represented in Table 1.

All neonates in group SR (n = 3) underwent initial surgery within the first 3 weeks of life. An SR strategy was chosen due to poor preoperative condition (n = 1), the presence of IAA, where prolonged CPB time is expected for complete repair (n = 1) or both (n = 1). Two patients were in poor clinical condition: 1 patient arrived in
<table>
<thead>
<tr>
<th>Group/PR</th>
<th>Case no.</th>
<th>Sex</th>
<th>Age (days)</th>
<th>Weight (kg)</th>
<th>Cardiac diagnosis</th>
<th>Extracardiac abnormalities</th>
<th>Postoperative hospital stay (days)</th>
<th>Surgery</th>
<th>Reoperation</th>
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<tbody>
<tr>
<td>PR</td>
<td>1</td>
<td>M</td>
<td>14</td>
<td>2.9</td>
<td>CAT-A1, dysplastic quadricuspid truncal valve with mild stenosis and regurgitation, PFO, right aortic arch, aberrant left subclavian artery</td>
<td>DiGeorge syndrome, primary hypoparathyroidism, s/p ICH II°, bilateral intracranial ventriculomegaly</td>
<td>33 None</td>
<td>Primary CAT repair with 14 mm Contegra valved RV-PA conduit</td>
<td></td>
</tr>
<tr>
<td>PR</td>
<td>2</td>
<td>M</td>
<td>30</td>
<td>4.1</td>
<td>CAT-A2, dysplastic quadricuspid truncal valve with mild stenosis and regurgitation, moderate mitral valve regurgitation</td>
<td>None</td>
<td>Primary CAT repair with 12 mm Contegra valved RV-PA conduit; truncal valve replacement (21 mm mechanical valved conduit)</td>
<td>18 Truncal valve replacement (21 mm mechanical valved conduit)</td>
<td></td>
</tr>
<tr>
<td>PR</td>
<td>3</td>
<td>M</td>
<td>30</td>
<td>3.8</td>
<td>CAT-A1, right aortic arch, multiple ASD II</td>
<td>Epilepsy</td>
<td>Primary CAT repair with 12 mm Contegra valved RV-PA conduit; delayed sternal closure</td>
<td>42 Pacemaker implantation, PA plasty, RV-PA conduit replacement</td>
<td></td>
</tr>
<tr>
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<td>4</td>
<td>M</td>
<td>54</td>
<td>3.3</td>
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<td>None</td>
<td>Primary CAT repair with 12 mm Contegra valved RV-PA conduit</td>
<td>14 None</td>
<td></td>
</tr>
<tr>
<td>PR</td>
<td>5</td>
<td>F</td>
<td>39</td>
<td>3.3</td>
<td>CAT-A1, anteriorly malaligned outlet septum, ASD II, PDA</td>
<td>None</td>
<td>Primary CAT repair with 12 mm Contegra valved RV-PA conduit; septal myectomy; delayed sternal closure</td>
<td>RV-PA conduit replacement</td>
<td></td>
</tr>
<tr>
<td>PR</td>
<td>6</td>
<td>M</td>
<td>34</td>
<td>3.7</td>
<td>CAT-A2, VSD, ASD II, right aortic arch, PA hypertension</td>
<td>DiGeorge syndrome, primary hypoparathyroidism</td>
<td>Primary CAT repair with 12 mm Contegra valved RV-PA conduit; delayed sternal closure</td>
<td>19 None</td>
<td></td>
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<tr>
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<td>F</td>
<td>50</td>
<td>3.6</td>
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<td>10 None</td>
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<tr>
<td>PR</td>
<td>8</td>
<td>F</td>
<td>50</td>
<td>3.4</td>
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<td>Primary CAT repair with 12 mm Contegra valved RV-PA conduit; delayed sternal closure</td>
<td>Truncal valve repair, PA plasty, RV-PA conduit replacement</td>
<td></td>
</tr>
<tr>
<td>PR</td>
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<td>M</td>
<td>19</td>
<td>3.6</td>
<td>CAT-A2, bicuspid truncal valve, VSD, ASD II, right aortic arch, flow induced PA hypertension</td>
<td>Sickle cell anemia</td>
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<td>11 None</td>
<td></td>
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<tr>
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<td>10</td>
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<td>46</td>
<td>3.9</td>
<td>CAT-A1, bicuspid truncal valve, VSD, ASD II, left lung hypoplasia</td>
<td>None</td>
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<td>Primary CAT repair with RV-PA conduit; truncal valve replacement (12 mm Hancock)</td>
<td></td>
</tr>
<tr>
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<td>11</td>
<td>F</td>
<td>91</td>
<td>4.3</td>
<td>CAT-A1, mesocardia, bicuspid truncal valve, VSD, ASD II, LPA hypoplasia, small MPA, supravalvar aortic stenosis, L-SVC, pentalogy of fallot, diaphragmatic defect</td>
<td>Psychomotor retardation, muscular hypotonia, plagiocephalus, post-pyloric feeding due to recurrent tracheal stenosis</td>
<td>Primary CAT repair with direct RV-PA anastomosis</td>
<td>Trachea slide plasty, redo trachea replacement, LA plasty replacement</td>
<td>167 Trachea slide plasty replacement; redo trachea replacement, LA plasty replacement</td>
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<td>None</td>
<td>Primary CAT repair with 12 mm Hancock valved RV-PA conduit</td>
<td>Primary CAT repair with RV-PA conduit replacement (12 mm Hancock)</td>
<td>16 None</td>
</tr>
<tr>
<td>PR</td>
<td>13</td>
<td>M</td>
<td>30</td>
<td>4.0</td>
<td>CAT-A1, mild truncal valve regurgitation, right aortic arch, PFO</td>
<td>None</td>
<td>Primary CAT repair with 10 mm Hancock valved RV-PA conduit</td>
<td>Primary CAT repair with RV-PA conduit replacement (10 mm Dacron valveless RV-PA conduit)</td>
<td></td>
</tr>
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<td>M</td>
<td>30</td>
<td>4.0</td>
<td>CAT-A1, mild truncal valve regurgitation, right aortic arch, PFO</td>
<td>None</td>
<td>Primary CAT repair with 10 mm Dacron valveless RV-PA conduit</td>
<td>Closure of residual VSD, RV-PA conduit replacement (12 mm Dacron valveless RV-PA conduit)</td>
<td></td>
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<tr>
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<td>M</td>
<td>30</td>
<td>4.0</td>
<td>CAT-A1, mild truncal valve regurgitation, right aortic arch, PFO</td>
<td>None</td>
<td>Primary CAT repair with 10 mm Dacron valveless RV-PA conduit</td>
<td>Trachea slide plasty, redo trachea replacement, LA plasty replacement</td>
<td></td>
</tr>
<tr>
<td>PR</td>
<td>16</td>
<td>M</td>
<td>30</td>
<td>4.0</td>
<td>CAT-A1, mild truncal valve regurgitation, right aortic arch, PFO</td>
<td>None</td>
<td>Primary CAT repair with 10 mm Dacron valveless RV-PA conduit</td>
<td>Trachea slide plasty, redo trachea replacement, LA plasty replacement</td>
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</tr>
<tr>
<td>PR</td>
<td>17</td>
<td>M</td>
<td>30</td>
<td>4.0</td>
<td>CAT-A1, mild truncal valve regurgitation, right aortic arch, PFO</td>
<td>None</td>
<td>Primary CAT repair with 10 mm Dacron valveless RV-PA conduit</td>
<td>Trachea slide plasty, redo trachea replacement, LA plasty replacement</td>
<td></td>
</tr>
<tr>
<td>PR</td>
<td>18</td>
<td>F</td>
<td>30</td>
<td>4.0</td>
<td>CAT-A1, mild truncal valve regurgitation, right aortic arch, PFO</td>
<td>None</td>
<td>Primary CAT repair with 10 mm Dacron valveless RV-PA conduit</td>
<td>Trachea slide plasty, redo trachea replacement, LA plasty replacement</td>
<td></td>
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<tr>
<td>PR</td>
<td>19</td>
<td>F</td>
<td>30</td>
<td>4.0</td>
<td>CAT-A1, mild truncal valve regurgitation, right aortic arch, PFO</td>
<td>None</td>
<td>Primary CAT repair with 10 mm Dacron valveless RV-PA conduit</td>
<td>Trachea slide plasty, redo trachea replacement, LA plasty replacement</td>
<td></td>
</tr>
</tbody>
</table>

ARSA reimplantation, 6 mm Gore-Tex

Closure of VSD and PFO, RV-PA shunt

RV-PA shunt conduit replacement

Group SR

16 VSD closure, RV-PA conduit replacement

CHARGE syndrome, bilateral cleft lip and palate, bilateral hearing impairment, hydrocephalus, multiple cerebral dysgenesis, epilepsy, renal pelvis dilatation

2 M 21 3.3 CAT-A1, bicuspid truncal valve, VSD, PFO, preoperative cardiogenic shock

3 M 7 2.8 CAT A4, IAA-B, bicuspid truncal valve, mild truncal valve regurgitation, VSD, ASD II

Follow-up was completed with a median duration of 3.6 years (range: 8 months to 11 years). There was neither early nor late mortality in both groups. In group SR, the median interval to second stage surgery was 216 days (range: 216–260 days). At this time, all patients underwent VSD closure and RV-PA conduit replacement. In 1 case, a truncal valve repair due to mild central regurgitation was simultaneously performed. There was no reoperation for recurrent aortic arch obstruction or recoarctation in group SR.

Seven patients (54%) in group PR required reoperation for RV-PA conduit failure (n = 4), truncal valve repair/replacement...
Within the immediate postoperative period after CAT repair, Hanley et al. [10] observed fewer pulmonary hypertensive episodes, lower pulmonary artery pressure and significantly shorter duration of ventilator dependence in children undergoing the operation before 30 days of age. In the present series, we observed 3 cases (23%) of postoperative pulmonary hypertensive crisis, managed effectively.

The progressive improvement in postoperative outcomes for patients with CAT, a continuing debate about the optimal timing of surgical repair remains [1, 2]. Some authors prefer elective surgical repair of CAT without major coexisting cardiovascular lesions at 1–3 months postnatally [2, 5, 6], whereas others advocate PR in the neonatal period [1, 7, 8]. Undoubtedly, in symptomatic neonates poorly controlled with medication, surgery should be performed as soon as possible. However, as pulmonary vascular resistance decreases within the first 2–3 weeks postnatally [9], it is our policy to repair straightforward CAT electively at the first to third month of life in patients without signs of congestion. Patients also typically grow and gain weight during this waiting period. Delayed surgery on ‘larger’ patients lowers the risk of neurological complications and enables ‘comfortable’ implantation of larger conduits, which might lead to longer freedom from reoperation. We are frequently able to implant at least 11 mm valved RV-PA conduits in young infants. Placement of much larger conduits is generally avoided due to the required larger ventriculotomy as well as the risk of coronary compression and PA distortion. In theory however, this policy might endanger patients with rapid development of congestive heart failure during the waiting period [2]. Therefore, careful surveillance is essential, even if the child is in a well-tolerated situation [2]. With careful follow-up, no mortality was noted during waiting period.

Further delaying repair beyond the first 2–3 months postnatally should not be recommended as this approach might increase the risk of pulmonary vascular disease, congestive heart failure and cardiopulmonary decompensation [6]. The cardiopulmonary deterioration could be explained by the presence of significant diastolic run-off into the low pressure pulmonary vasculature, away from coronary and systemic perfusion. The restricted coronary perfusion and the persistent excessive ventricular volume load might further deteriorate the overall myocardial performance. Taking all these aspects into account, outcome is favourably affected by the presence of PA obstructive lesions. In our series, 3 patients in group PR presented initially with hypoplastic PAs. They underwent complete repair within 46–91 days postnatally.

DISCUSSION

Simple common arterial trunk

Despite the progressive improvement in postoperative outcomes for patients with CAT, a continuing debate about the optimal timing of surgical repair remains [1, 2]. Some authors prefer elective surgical repair of CAT without major coexisting cardiovascular lesions at 1–3 months postnatally [2, 5, 6], whereas others advocate PR in the neonatal period [1, 7, 8]. Undoubtedly, in symptomatic neonates

(n = 2) or both (n = 1). Median interval to second surgery was 1 year (range: 5 months–8 years). RV-PA conduit replacement was necessary due to conduit stenosis (n = 1), conduit regurgitation (n = 2) and RV dilatation after previous implantation of a valveless conduit (n = 1). One valved conduit was secondarily implanted in a patient with free pulmonary regurgitation after direct RV-PA anastomosis at initial repair. Kaplan–Meier freedom from reoperation after 1, 2 and 8 years was 77 ± 12, 68 ± 13 and 20 ± 17% in group PR. Correspond to the staged strategy, all patients in group SR required reoperation to complete the anatomical correction (log-rank P < 0.01, Fig. 2A). In group SR however, there was no surgical reintervention of truncal valve, aortic arch or RV-PA conduit after the second stage procedure (Fig. 2B).

Three truncal valve procedures (one valve repair and two valve replacements) were performed due to severe truncal valve regurgitation. In 2 cases, initial preoperative echocardiography showed moderate truncal valve regurgitation. Another patient presented primarily with a dysplastic truncal valve and moderate stenosis. Intraoperative inspection showed an acceptable truncal valve function and antegrade cardioplegia was considered adequate. Therefore, we decided not to perform truncal valvuloplasty at initial surgery. Over time, patients developed worsening truncal valve regurgitation, necessitating a truncal valve repair or replacement 5 months, 7 months and 8 years after initial surgery.

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with inhaled nitric oxide. All cases were repaired primarily at 30, 34 and 50 days postnataally. Ventilation arrangement that kept arterial PaCO2 at about 30 mmHg, arterial PaO2 above 120 mmHg and arterial pH at 7.50 as well as controlled use of sedatives and muscle relaxants help to reduce the tendency for pulmonary vascular resistance to rise. This experience in preventing pulmonary hypertensive episodes along with the availability of inhaled nitric oxide and iloprost has effectively reduced perioperative morbidity.

Complex common arterial trunk

For children with CAT and major associated cardiovascular lesions, such as moderate to severe truncal valve regurgitation and hypoplastic or IAA, early complete repair has been widely preferred [11, 12]. Although 12.5% hospital mortality can be achieved in experienced centres [12, 13], a multi-institutional study published in 2006 by the Congenital Heart Surgeons Society reported a 68% mortality rate after single stage repair of CAT with IAA [14]. Postoperative low cardiac output was responsible for the majority of deaths in this study [14]. In 2012, the Society of Thoracic Surgeons reported that the combination of IAA and CAT was associated with 28% mortality [3]. Even though some authors recently bring this association into question [7, 15], it is unrealistic to conclude that such complex cases are no longer challenging. Miyamoto et al. [16] reported 50% hospital mortality after CAT–IAA repair, mainly in patients with ACC score of 18 or greater; only 1 patient with ACC score greater than 18 survived the surgery. In our series, mean ACC score was 11 ± 0.6 (range: 11–13) in group PR and 18 ± 3.1 (range: 15–21) in group SR (P < 0.01). As hospital mortality correlates with ACC score, our institution favours a staged strategy in high-risk cases, similar to what has been as described by Sernich et al. [4].

Initial repair step by separating systemic and pulmonary circulation and elimination of aortic arch obstruction at an early age avoids the diastolic run-off away from coronary perfusion and reduces LV volume load. The small (6 mm) RV-PA shunt limits the pulmonary blood flow sufficiently, resulting in well-balanced the ratio of pulmonary to systemic flow without any requirement for perioperative intervention. It might cause limited pulmonary reperfusion during postoperative transient pulmonary hypertensive crisis; however, this is typically well tolerated in the presence of VSD. In addition, the small shunt diameter reduces the size of the required right ventriculotomy in delicate neonatal hearts with fairly compromised function [4]. Compared with the complete PR, staged approach also shortens the myocardial ischaemic and CPB time. These aspects are critical as the implication of intraoperative ischaemia is potentiated in neonates with compromised myocardial function. Myocardial dysfunction might be partly caused by preoperatively reduced diastolic coronary flow and limited subendocardial coronary perfusion, which is triggered by the volume-overload induced elevation of left ventricular end diastolic pressure.

At the second stage, a ventriculotomy site that accommodates a larger RV-PA conduit provides excellent access to VSD closure. When compared with VSD closure during the first surgery, our staged strategy might limit the risk of truncal valve injury, intracardiac trauma and RV dysfunction in sensitive neonatal hearts. Moreover, leaving the VSD open can be beneficial as it reduces the risk of low cardiac output caused by transient RV dysfunction following ventriculotomy and shunt placement. It further reduces the haemodynamic impact of postoperative pulmonary hypertensive crises. In the present series, no postoperative low cardiac output was observed. One patient showed transient episodes of desaturation postoperatively (Case 2, group SR). This might be caused by right-to-left shunt across the VSD. However, the role of pulmonary hypersecretion on desaturation episodes (as observed preoperatively) could not be completely excluded. As described by Sernich et al. [4] leaving a VSD along with an RV-PA shunt might cause RV volume overload. They reported that no adverse haemodynamic consequences had been observed postoperatively. Similarly, we did not observe postoperative RV volume overload after SR. In our experience, SR strategy seems to be associated with stable postoperative course and improved hospital survival. This upfront mortality is by far the most urgent issue, as the second stage surgery is usually safe to perform. The last aspect is in agreement with low mortality after CAT reoperation reported from other centres [12, 17]. All CAT patients survive reinterventions and reoperations performed on RV-PA conduit, truncal valve, pulmonary arteries and airway obstruction [11, 17].

Truncal valve procedure

Preoperative evaluation of truncal valve function and anatomy typically indicates some degree of obstruction due to high blood flow across the valve, which comprises both systemic and pulmonary flow. This increased amount of blood flow could be further exaggerated if truncal valve regurgitation is present. Thus, a preoperative high pressure gradient across the truncal valve does not necessarily translate into significant stenosis [2]. In our series, no true primary truncal valve stenosis was observed.

In contrast, truncal valve regurgitation requiring surgery at the time of CAT repair has been reported as a risk factor for postoperative mortality [3]. This outcome might be related to the interference with intraoperative myocardial protection and unfavourable postoperative haemodynamic consequence. However, the grade of truncal valve regurgitation that required repair is difficult to assess preoperatively. The pulmonary run-off might mask the severity of regurgitation. On the other hand, truncal valve regurgitation might decrease postoperatively and it is usually well tolerated [2]. This might be associated with the reduction of truncal valve annular dilatation parallel to the reduction of LV volume overload [2]. In our series, less than moderate degree of truncal valve regurgitation was left untouched. In all cases, truncal valve function was inspected and tested under direct vision. The adequacy of antegrade cardioplegia was observed upon cardioplegia arrival to the coronary sinus. Furthermore, as haemodynamic impact of truncal valve regurgitation is intensified in the presence of residual aortic obstruction, establishing an unobstructed aortic arch is very important.

Limitations

The present analysis shares the limitations of a retrospective review undertaken in a non-randomized uncontrolled patient population with a varying duration and intensity of follow-up. Another limitation is the small number of study patients that might not be enough to pick up a statistically significant difference. These aspects might limit the validity of the results.

CONCLUSION

Routine elective CAT repair could be safely performed at 1–3 months of age. However, neonatal CAT repair could be associated
with a higher mortality especially in the presence of IAA. In such cases, a staged CAT repair seems to be associated with favourable postoperative course and improved hospital survival, despite the inevitable requirement for reoperation, which can be performed at relatively low risk.

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**REFERENCES**