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ORIGINAL ARTICLE

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Anomalous left coronary artery from pulmonary artery repair: Outcomes from the European Congenital Heart Surgeons Association Database

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Abstract

Introduction: We sought to determine the surgical outcomes of patients with anomalous origin of the left coronary artery from the pulmonary artery (ALCAPA) enrolled in the European Congenital Heart Surgeons Association (ECHSA) database.

Materials and Methods: From 1999 to 2019, 907 patients with ALCAPA underwent surgical repair and were included in the current study. The primary outcome was in-hospital mortality. Secondary outcomes included frequency and results of concomitant mitral valve surgery and postoperative mechanical circulatory support (MCS).

Results: The overall in-hospital mortality was 6% (54/907) and was significantly higher in neonates ($p = .01$), patients with lower body surface area (BSA) ($p = .01$), and those requiring postoperative MCS ($p = .001$). Associated mitral valve surgery was performed in 144 patients (15.9%) and was associated with longer cardiopulmonary bypass (CPB) and aortic cross-clamp times (AOX) ($p \leq .0001$) but was not

significantly related to an increase in in-hospital mortality. Postoperative MCS was required in 66 patients (7.3%). These patients were younger ($p \leq .001$), had a lower BSA ($p \leq .001$), and required a longer CPB ($p \leq .001$) and AOX time ($p \leq .001$).
Conclusions: ALCAPA repair can be achieved successfully, and with low surgical risk. Concomitant mitral valve procedures can be performed without increasing operative mortality. The use of MCS remains a valuable option, especially in younger patients.

KEYWORDS
congenital heart disease, coronary arteries anomalies, surgical outcomes

1 | INTRODUCTION

Anomalous origin of the left coronary artery from the pulmonary artery (ALCAPA) is a rare congenital heart abnormality affecting 1 in 300,000 live births.[1] The anomaly is usually isolated but is occasionally associated with other congenital cardiac defects such as patent ductus arteriosus, ventricular septal defect, tetralogy of Fallot, or coarctation of the aorta. If left untreated, the mortality can reach up to 90% within the first year of life. Direct transfer and reimplantation of the anomalous coronary artery into the aorta is the preferred surgical technique, when feasible. In the case of unfavorable coronary anatomy, an intrapulmonary artery baffle, originally introduced by Takeuchi and colleagues, can be performed.[2] Coronary artery bypass grafting may be a valuable option, especially in adult patients with ALCAPA, where reimplantation may be more difficult because of increased coronary artery friability and lower vessel elasticity for mobilization.

Mitral valve (MV) surgery at the time of ALCAPA repair is a controversial issue. Many centers do not perform mitral valve repair, claiming that mitral valve insufficiency (MVI) usually improves after coronary reimplantation, especially in infants.[3–6] Others advocate routine MV repair regardless of the degree of MVI, with the purpose of improving cardiac output.[7,8]

In patients who cannot be weaned from cardiopulmonary bypass (CPB), mechanical circulatory support (MCS), such as extracorporeal membrane oxygenation (ECMO) or left ventricle assist devices (LVAD) can be a valuable option that allows severely damaged ventricles to decompress and recover after ALCAPA repair or as a bridge to heart transplantation.[9–12]

In the current study, we sought to analyze data from the European Congenital Heart Surgeons Association (ECHSA) database to determine surgical outcomes of ALCAPA repair, frequency, and results of concomitant MV surgery, and need for postoperative MCS.

2 | MATERIALS AND METHODS

2.1 | Data source

A retrospective analysis of data from the ECHSA database was performed. The procedures followed were in accordance with recording review and protection of patient confidentiality. The ECHSA Scientific Committee approved the study. The ECHSA database gathers deidentified surgical data on 300,000 procedures from 403 congenital cardiothoracic surgical centers in 86 countries, 70% of the data comes from European centers and 30% from non-European centers. Data are submitted into the database using the International Pediatric and Congenital Cardiac Code in the version of the International Congenital Heart Surgery Nomenclature and Database Project of the European Association for Cardio-Thoracic Surgery and the Society of Thoracic Surgeons. Detailed relevant information is available on the ECHSA database and has already been published.[13–15]

2.2 | Patient population

All the patients with a diagnosis coded as DIAG159, "Coronary artery anomaly, anomalous pulmonary origin (includes ALCAPA)" and DIAG094, "Anomalous origin of the coronary artery from pulmonary artery" were enrolled (Figure 1). Of the initial pool of patients reviewed, 907 unique cases of ALCAPA underwent repair from 1999 to 2019. Patients with a diagnosis of anomalous aortic origin of the coronary artery (AAOCA) were excluded from the analysis, as well as patients with associated complex cardiac anomalies (mainly hypoplastic left heart syndrome and transposition of the great arteries) and patients with incomplete demographical data or a mismatch between diagnosis and procedure performed. All patients included in the study underwent ALCAPA repair (code "PROC117"), with or without concomitant procedures. ALCAPA repair was arbitrarily defined as "simple" in all cases where ALCAPA was an isolated diagnosis or when associated diagnoses of MVI, patent ductus arteriosus, and patent foramen ovale or atrial septal defect were present. When the diagnosis of ALCAPA was associated with other congenital heart defects (ventricular septal defects, valve defects other than MVI, tetralogy of Fallot, atrioventricular canal, abnormal pulmonary venous return, cor triatriatum, and Shone complex) the term "complex" ALCAPA repair was applied. In the ECHSA database, there are no separate codes for direct reimplantation or Takeuchi procedure, whereas coronary artery bypass grafting has the code "PROC127," which was also included in the search (four patients fell into this category, three adults and one newborn).

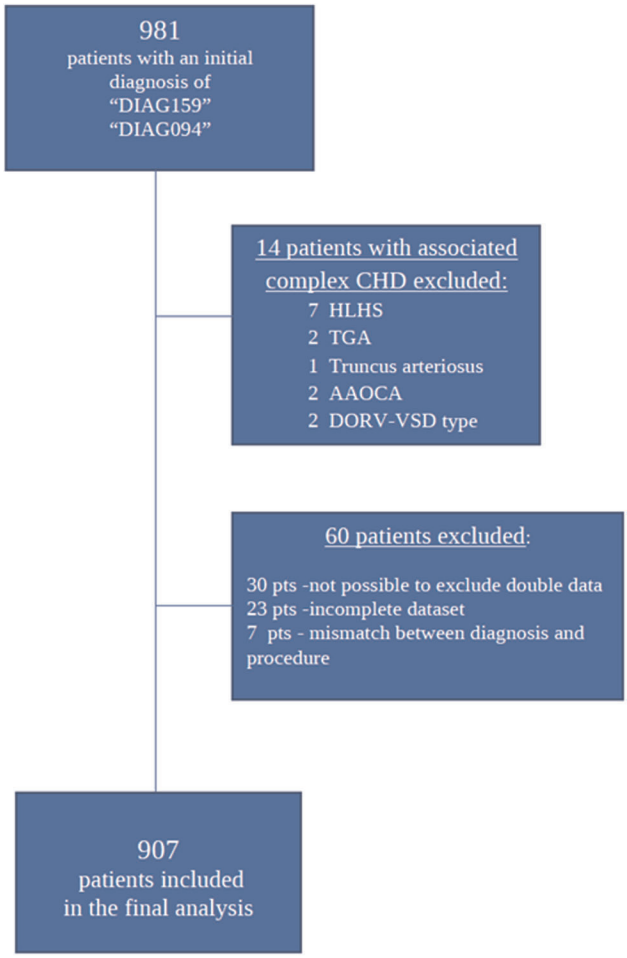

FIGURE 1 Consort diagram of the study. AAOCA, anomalous aortic origin of a coronary artery; CHD, congenital heart disease; DIAG094, anomalous origin of the coronary artery from a pulmonary artery; DIAG159, coronary artery anomaly, anomalous pulmonary origin; DORV-VSD type, double outlet right ventricle, ventricular septal defect type; HLHS, hypoplastic left heart syndrome; TGA, transposition of the great arteries

Demographic and operative variables were analyzed. The primary outcome was in-hospital mortality. Secondary outcomes were the frequency and results of concomitant MV surgery as well as the need for postoperative MCS.

2.3 | Statistical analysis

Variables are expressed as a median (interquartile range) when continuous, or as a number (percentage) when categorical. To compare various groups and determine correlations among variables, the Wilcoxon rank-sum was used for continuous variables and the Fisher's exact test was used for dichotomous variables. Univariate analysis was performed to determine the association between variables and in-hospital mortality. The probability of type I error was set at $\alpha = .05$. We analyzed data with the R-System statistical package, using Harrell's rms libraries.[16]

3 | RESULTS

3.1 | Population and operative characteristics

Demographics and characteristics of the overall population are shown in Table 1. Of the 907 patients included, 40.1% ($n = 364$) were male, and 63.4% ($n = 575$) were aged 1 year old. Most of the procedures (88.9%, $n = 806$) were performed in European centers, and similarly, the majority of these procedures (70.6%; $n = 640$) were performed in the second decade of this study period (Figure 2). When comparing between survivors and nonsurvivors, patient BSA (0.33, 0.28–0.58 vs. 0.30, 0.27–0.36 m^2; $p = .01$), and preoperative weight (6.30, 5–13 vs. 5.60, 4.6–7.2 kg; $p = .003$) were predictive for in-hospital mortality.

Operative characteristics associated with in-hospital mortality included CPB time (131, 101–164 vs. 144, 110–221 min; $p = .046$) and intermittent pulmonary pressure ventilation (IPPV) time (65, 17–162 vs. 144, 28–608 min; $p = .007$). Postoperative MCS was also associated with in-hospital mortality. LVAD implantation was performed in 24 patients, 19 (2.2%) survived and 5 (9.3%) experienced in-hospital mortality ($p = .002$). Similarly, ECMO was performed in 44 patients, 35 (4.1%) survived and 9 (16.7%) died in-hospital ($p < .001$).

3.2 | Operative characteristics by age group

Analysis of patient characteristics and outcomes was performed according to patient age groups and is summarized in Table S1. Between the four age groups (neonates ≤ 30 days vs. infants = 1–12 months vs. older children = 1–18 years vs. adults ≥ 18 years), lower weight and BSA were significantly associated with higher mortality ($p = .003$ and $p = .01$, respectively). There was no difference between surgical eras.

Median CPB time was 132 (101–165) min and median aortic cross-clamp time (AOX) was 70 (52–92) min. Of the 907 patients undergoing ALCAPA repair, 54 (6%) experienced in-hospital mortality. Mortality rates were 25% among neonates, 6.4% in infants, 4.6% in older children (1–18 years), and 1.8% in adults. The majority of LVAD ($n = 23/24$) and ECMO ($n = 43/44$) implantations were performed in infants, with one instance of each in older children. Similarly, all the instances of delayed sternal closure ($n = 16$) were performed in infants. The infant cohort also had a longer median length of stay in the intensive care unit (ICU) ($n = 10$ days, 5–16) and hospital length of stay ($n = 17$ days, 12–27) compared to the entire population and all other age groups ($p < .001$ and $p < .001$, respectively).

3.3 | Mitral valve interventions by age group

Characteristics and operative outcomes for patients receiving concomitant MV surgery are shown in Tables S1 and S2. In 144 patients (15.9%) a MV procedure was performed at the time of ALCAPA repair. The most common procedures included MV plasty ($n = 132$) and MV replacement ($n = 5$). Patients receiving MV surgery were overall older (median 10, 4–38.5 months; $p = .001$), almost half MV

TABLE 1 Patient's characteristics according to hospital outcome

Demographics	All patients	Survivors	Nonsurvivors	*p* value
No. of patients[a]	907	853 (94)	54 (6)	
Sex (males)[a]	364 (40.1)	340 (39.6)	24 (44.4)	.51
Age (months)[b]	6 (3–29)	6 (3–35)	4 (3–10)	.06
<30 days[a]	16 (1.8)	12 (1.4)	4 (7.4)	
1–12 months[a]	575 (63.4)	538 (63.1)	37 (68.5)	
1–18 years[a]	259 (28.6)	247 (29)	12 (22.2)	
>18 years[a]	57 (6.3)	56 (6.6)	1 (1.9)	
BSA (m^2)[b]	0.33 (0.28–0.57)	0.33 (0.28–0.58)	0.30 (0.27–0.36)	.01
Weight (kg)[b]	6.20 (5–12)	6.30 (5–13)	5.60 (4.6–7.2)	.003
Surgical era				.21
1999–2008[a]	267 (29.4)	247 (29)	20 (37)	
2009–2019[a]	640 (70.6)	606 (71)	34 (63)	
Operative and outcomes				
Simple ALCAPA repair[a]	861 (94.9)	811 (95.1)	50 (92.6)	.42
CPB (min)[b]	132 (101–165)	131 (101–164)	144 (110–221)	.046
AOX (min)[b]	70 (52–92)	70 (52–92)	75 (64–96)	.089
IPPV time (h)[b]	67 (18–168)	65 (17–162)	144 (28–608)	.007
MV procedure[a]	144 (15.9)	135 (15.8)	9 (16.7)	.435
MV valvuloplasty[a]	132	125	7	
MV valve replacement[a]	5	4	1	
MV, others[a]	7	6	1	
LVAD implantation	24 (2.6)	19 (2.2)	5 (9.3)	.002
ECMO[a]	44 (4.9)	35 (4.1)	9 (16.7)	<.001
Delayed sternal closure[a]	16 (1.8)	15 (1.8)	1 (1.9)	.965
ICU length of stay (days)[b]	6 (2–13)	6 (2–13)	9 (3–25)	.078
LOS (days)[b]	14 (8–22)	14 (8–22)	12 (3–24)	.037
Hospital mortality[a]	54 (6)	0	54	<.001

Abbreviations: AOX, aortic cross-clamp time; ALCAPA, anomalous origin of the left coronary artery from a pulmonary artery; BSA, body surface area; CPB, cardiopulmonary bypass; ECMO, extracorporeal membrane oxygenation; ICU, intensive care unit; IPPV, intermittent pulmonary pressure ventilation; LOS, hospital length of stay; LVAD, left ventricular assist device; MV, mitral valve

[a]Number of patients and percentage.

[b]Median and interquartile range.

repairs (47%) were performed in children aged 1 year or older. The majority of concomitant MV procedures ($n = 115$, 79.9%) were performed in the second half of the study period ($p = .008$). Concomitant MV surgery increased CPB time (141, 118–172 vs. 130, 98–164 min; $p < .001$) and AOX time (89, 73–112 vs. 65, 49–87 min; $p < .001$). However, this was not associated with higher mortality.

3.4 | MCS by age group

Postoperative MCS was required in 66 (7.3%) patients, 97% ($n = 64$) were infants ($p < .001$). The majority of MCS was performed in European centers ($n = 51$, 77.3%; $p = .002$) and in the second half of the study period ($n = 61$, 92.4%; $p < .001$). Patients that required MCS had significantly longer CPB (167, 140–225 vs. 128, 99–162 min; $p < .001$) and AOX times (80, 65–109 vs. 69, 51–91; $p < .001$). These patients required longer IPPV time (315, 206–442 vs. 48, 14–144 h; $p < .001$), as well as longer ICU (19, 12–35 vs. 6, 2–12 days; $p < .001$) and hospital length of stay (29, 20–46 vs. 13, 8–20 days; $p < .001$). Concomitant MV procedures were not associated with the need for postoperative MCS. In-hospital mortality was observed in 14 MCS cases (21.2%). Postoperative MCS was associated with a higher mortality ($p < .001$ for ECMO and $p = .002$ for LVAD). A complete summary of patients' characteristics and outcomes according to the need for MCS is provided in Table S3.

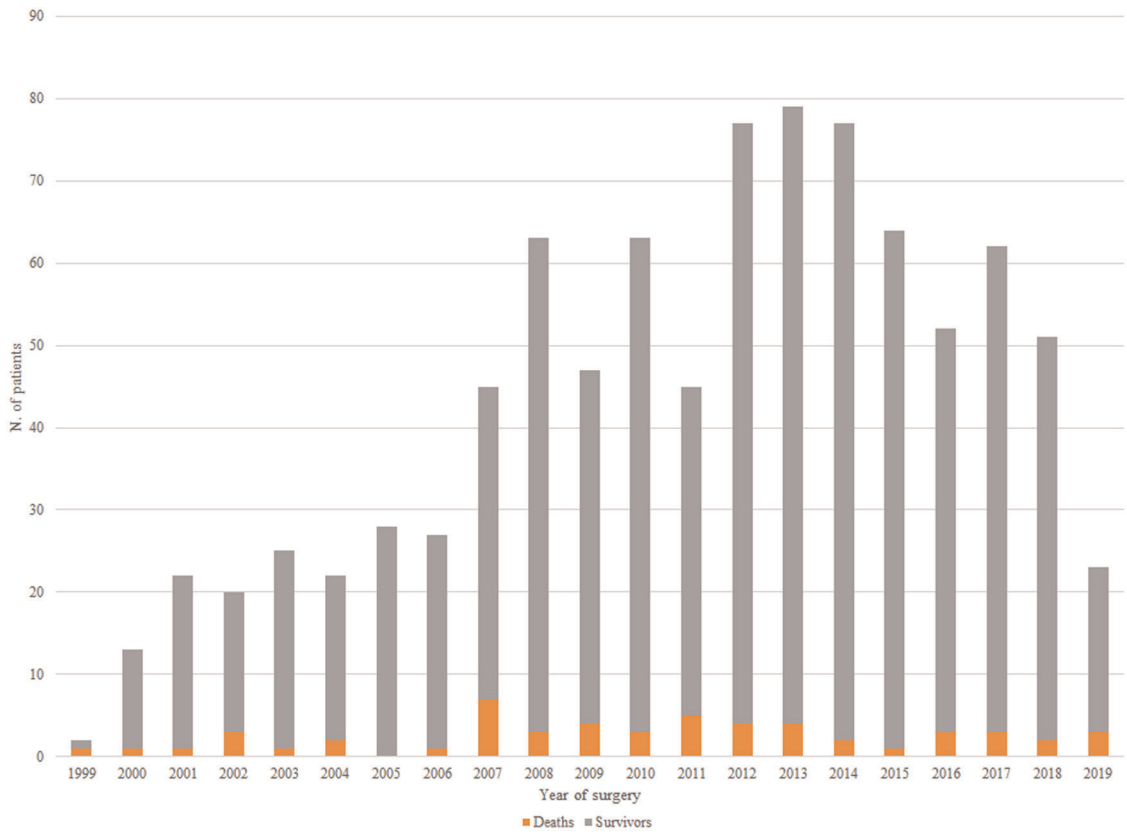

FIGURE 2 Histogram illustrating the timeline of the outcomes and trends of anomalous origin of the left coronary artery from the pulmonary artery repair within the European Congenital Heart Surgeons Association Database

4 | DISCUSSION

ALCAPA represents the most common cause of myocardial ischemia and infarction in children.[1] Closure of the ductus arteriosus and the decrease in pulmonary vascular resistance creates a "coronary steal" phenomenon with left-to-right shunting from the coronary circulation into the pulmonary artery leading to myocardial ischemia. The development of inter-coronary collateral vessels from the right coronary artery to the left coronary arterial system may mitigate this process. Infants typically present with signs and symptoms of congestive heart failure, important left ventricular dysfunction, and MVI, due to little or no coronary collateral development. In adults, in whom sufficient collaterals developed over time, typical presentation includes malignant arrhythmias and sudden death related to ischemia usually present around 35 years of age.

Surgical restoration of a dual coronary artery system, either by direct aortic reimplantation or the Takeuchi procedure,[2] is the gold-standard for treatment of ALCAPA as soon as the diagnosis is established. ALCAPA repair carries a surgical mortality risk that ranges from 0% to 23%.[1] As reported by other authors, there is no significant difference between the various surgical techniques, except for simple ligation.[17] In our large multicenter cohort study, the mortality was low (6%) (Figure 2), comparable to that of the most recent series, but still double that of the STS Congenital Database (2.8%)[18] and the United Kingdom database (2.5%).[19,20] Although there were no significant differences in the patient population, surgical variables, or the rates of the percentage of ECMO, the discrepancy we encountered could be related to the higher mortality found in neonates (25% in our series vs. 5.3% of Straka et al.).[18] As shown by other authors, in our study neonates ($p = .01$), lower body weight ($p = .003$), lower BSA ($p = .01$), and the need for postoperative MCS ($p < .001$) were all associated with higher in-hospital mortality.[1,18] We speculate that the need for longer CPB time (especially in neonates, who usually have less inter-coronary collaterals and may have sustained more extensive left ventricular damage) causes a more severe clinical presentation in our patients. Of note, in our neonatal group, no concomitant MV repairs were performed and ECMO support was not used.

MCS in pediatric patients after cardiac surgery can provide adequate systemic flow and decompression of the damaged ventricle (s), thereby contributing to the recovery of ventricular function.[9,10] In our series, postoperative MCS was needed in 66 (7.3%) patients, mostly infants, with lower weight and lower BSA. In particular, 44 patients received ECMO support and 24 implantations of LVAD. Overall mortality in the MCS group was 21.2% (20.5% for the ECMO group and 20.8% for LVAD). In accordance with the literature, the use of MCS was related to longer CPB time, AOX time, and longer duration of IPPV. Delayed sternal closure, longer ICU length of stay, and overall hospital length of stay were also more common in the

MCS group.[9–11] The need for postoperative MCS correlated with higher in-hospital mortality ($p < .001$ and $p = .002$ for ECMO and LVAD, respectively). Several studies have reported comparable results with the use of ECMO support.[10,11,21] Moreover, Del Nido et al. reported good long-term recovery of left-ventricular function with the use of LVAD after ALCAPA repair.[9] Indeed, patients with severe dysfunction of the left ventricle who cannot be weaned from CPB, may benefit from immediate MCS.

MVI is very common in patients with ALCAPA. There are different proposed mechanisms, such as left ventricle myocardial infarction, scarring and calcification of papillary muscles, papillary muscle displacement due to left ventricle dilatation, and endocardial fibroelastosis involving the whole MV apparatus. Many centers do not recommend intervening on the MV at the time of ALCAPA repair because of the prolonged AOX time in the context of an already severely compromised left ventricle but also because of the well-known improvements in MV function as early as few months after ALCAPA repair without intervention.[3–6] Other authors advocate routine MV surgery at the time of ALCAPA repair, to improve early postoperative cardiac output.[7,8] Alsoufi et al.[22] and more recently Weixler et al.[20] recommended concomitant MV repair in patients with severe MVI or in whom preoperative organic, rather than functional, MV pathology is diagnosed. In older children with important MVI, MV repair at the time of coronary reimplantation may be advisable because, unlike in infants, the cause of MVI could be irreversible myocardial or papillary damage, which is not expected to improve with recovery of left ventricular function.[23] Lastly, Huddleston et al.[3] recommended cardiac catheterization in patients with recurrent or persistent MVI after ALCAPA repair, to evaluate the patency of the reimplanted coronary artery before MV repair. In our cohort, MV repair was performed in 144 patients (15.9%). The majority of the patients received MV surgery later in their life and not during the index operation, a pattern that is borne out in the prior literature.[22] Concomitant MV surgery increased CPB time ($p < .001$) and AOX time ($p < .001$), but was not associated with higher in-hospital mortality. In fact, the additional ischemic time necessary for MV repair does not appear to have a negative impact on outcomes.[7,21] The ECHSA Congenital Database, being a registry with predefined data fields for all types of congenital disorders, does not include preoperative details nor the specifics of such interventions, which would have allowed us to discern the indications used by the various centers for concomitant MV intervention. Therefore, a word of caution is needed. Our data cannot support routine MV repair, but we have been able to demonstrate that in those selected cases where it must be performed (i.e., older children), MV surgery can be performed without increasing hospital mortality.

4.1 | Limitations

Our study has several limitations. This is a retrospective data examination over a long period of time and therefore both inter-institutional and intrainstitutional variability of surgical treatment was expected. Furthermore, our analysis was limited to in-hospital mortality and it was not possible to provide long-term follow-up data. The ECHSA database collects data from countries with different social and economic backgrounds, therefore differences in access to medical care may also have influenced the results (e.g., preoperative conditions, clinical status, and timing at presentation). Considering that the study was restricted to the analysis of the standard minimal data set encoded in the registry, applicable to all congenital cardiac disorders, it was not possible to assess important clinical variables, such as preoperative left ventricle ejection fraction, which could have influenced surgical outcomes, and adequately determine risk factors for mortality and need for MCS. Similarly, the degree and characteristics of MV dysfunction were not available, and this would have enabled an analysis of the indications for concomitant mitral intervention. Nonetheless, we were able to contribute, with the largest data set to date, to the definition of the overall surgical risk for ALCAPA repair.

In conclusion, ALCAPA repair can be successfully performed with low surgical risk. Concomitant MV surgery can be performed safely without increasing in-hospital mortality. The need for MCS is associated with higher mortality but remains a fundamental option for patients with severe postoperative instability.

CONFLICT OF INTERESTS

The authors declare that there are no conflict of interests.

AUTHOR CONTRIBUTIONS

Drafting article: Laura Torlai Triglia, Alvise Guariento, and David Blitzer.*Critical revision of the article*: Lorenza Zanotto, Massimo Padalino, and Giovanni Di Salvo. *Data analysis/interpretation*: Lucia Zanotto and Renjie Hu. *Data analysis/interpretation; critical revision of the article*: Claudia Cattapan. *Approval of article*: Haibo Zhang. *Concept/design; critical revision of the article; approval of the article*: Claudia Herbstand Jurgen Hörer.*Critical revision of the article; approval of the article*: George Sarris and Tjark Ebels. *Data collection, approval of the article*: Bohdan Maruszewski and Zdzislaw Tobota. *Statistics*: Giulia Lorenzoni, Daniele Bottigliengo, and Dario Gregori. *Concept/design, approval of the article*:Vladimiro L. Vida.

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SUPPORTING INFORMATION

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