Early and long-term outcomes of aortic coarctation repair: a prospective two-center study

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Received: 19 January 2026. Revised: 26 January 2026. Accepted 28 January 2026.

How to cite: Teplov P.V., Miller A.Yu., Gvozd E.M., Polyakova Yu.N., Sakovich V.A., Amansakhatova E.N., Soynov I.A. Early and long-term outcomes of aortic coarctation repair: a prospective two-center study. *Patologiya krovoobrashcheniya i kardiokhirurgiya = Circulation Pathology and Cardiac Surgery*. 2026;30(1):26-39. (In Russ.)https://doi.org/10.21688/1681-3472-2026-1-26-39

Informed consent
The patient's official representative has provided informed consent for the use of the recordings for medical purposes.

Funding
The research was carried out within the state assignment of Ministry of Health of Russian Federation (No. 124022500251-0).

Conflict of interest
The authors declare no conflict of interest.

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Abstract

Introduction. Recoarctation of the aorta, arterial hypertension and main bronchus compression remain the major complications following surgical correction of aortic coarctation and aortic arch hypoplasia, despite high effectiveness and low mortality in most patients.

Objective. To evaluate early and long-term outcomes of surgical treatment in neonatal patients with aortic coarctation, comparing native tissue repair (such as extended end-to-end anastomosis) to patch aortoplasty techniques.

Methods. This two-center, prospective, randomized study analyzed the outcomes of surgical repair for aortic coarctation with arch hypoplasia in a pediatric patient cohort, which included 105 neonates. According to the study design, all patients were divided into two groups based on the surgical technique used: patch aortoplasty (70 patients) and repair with an extended end-to-end anastomosis (35 patients).

Results. In-hospital mortality in the patch repair group was 5 (7.1 %) patients, compared to 3 (8.6 %) patients in the native tissue repair group, $p > 0.999$. The only independent risk factor for mortality, regardless of the surgical technique performed, was the duration of cardiopulmonary bypass (OR with 95 % CI 1.374 to 45.242, $p = 0.016$). The sole predictor for the development of aortic recoarctation in the long-term postoperative period was a peak gradient across the isthmus exceeding 14 mm Hg (HR = 3.75; $p < 0.001$). The use of the patch repair surgical strategy was associated with a reduced risk of developing arterial hypertension (HR = 0.385, $p = 0.046$).

Conclusion. Both common surgical strategies demonstrate comparable efficacy in treating coarctation and hypoplasia of the aortic arch in newborns. Regardless of the surgical approach, primary attention should be paid to the individual anatomical features of the aortic arch, patient condition, and associated congenital heart defects. However, in all patients, complete resection of ductal tissue is mandatory to prevent the development of aortic recoarctation and residual arterial hypertension.

Keywords: aortic arch hypoplasia; aortic coarctation, aortoplasty; congenital heart disease; pediatric cardiology; pulmonary homograft

Ранние и отдаленные результаты коррекции коарктации аорты: проспективное рандомизированное двухцентровое исследование

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Поступила в редакцию 19 января 2026 г. Исправлена 26 января 2026 г. Принята к печати 28 января 2026 г.

Цитировать: Теплов П.В., Миллер А.Ю., Гвоздь Е.М., Полякова Ю.Н., Сакович В.А., Амансахатова Е.Н., Сойнов И.А. Ранние и отдаленные результаты коррекции коарктации аорты: проспективное рандомизированное двухцентровое исследование. *Патология кровообращения и кардиохирургия*. 2026;30(1):26-39. https://doi.org/10.21688/1681-3472-2026-1-26-39

Информированное согласие

Получено информированное согласие официальных представителей пациентов на использование медицинских данных в научных целях.

Финансирование

Работа выполнена в рамках государственного задания Министерства здравоохранения Российской Федерации (№ 124022500251-0).

Конфликт интересов

Авторы заявляют об отсутствии конфликта интересов.

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Аннотация

Актуальность. Рекоарктация аорты, артериальная гипертензия и компрессия главного бронха остаются основными осложнениями после хирургической коррекции коарктации аорты и гипоплазии дуги аорты, несмотря на низкую летальность и высокую эффективность у большинства пациентов.

Цель. Оценить ранние и отдаленные результаты хирургического лечения новорожденных пациентов с коарктацией аорты с использованием пластических операций нативными тканями в сравнении с аортопластикой заплатными методами.

Методы. В представленном многоцентровом проспективном рандомизированном исследовании выполнен анализ результатов хирургической коррекции коарктации с гипоплазией дуги аорты у педиатрической когорты пациентов, в исследование было включено 105 новорожденных. Согласно дизайну исследования в зависимости от способа хирургической коррекции все пациенты разделены на две группы: пластика дуги аорты с использованием заплаты (70 пациентов) и аортопластика с помощью косого расширенного анастомоза (35 пациентов).

Результаты. Госпитальная летальность в группе пластики с применением заплаты составила 5 (7,1 %) пациентов, в то время как в группе пластики нативными тканями – 3 (8,6 %) пациента, $p > 0,999$. Единственным фактором риска летального исхода, независимо от применяемой хирургической методики, являлась продолжительность искусственного кровообращения (95 % ДИ [1,374; 45,242]; $p = 0,016$). Единственным предиктором развития рекоарктации аорты в отдаленном послеоперационном периоде является пиковый градиент давления на перешейке более 14 мм рт. ст. (ОР = 3,75; $p < 0,001$). Использование хирургической тактики с использованием заплаты снижает риск развития артериальной гипертензии (ОШ = 0,385; $p = 0,046$).

Заключение. Обе распространенные хирургические стратегии демонстрируют сопоставимую эффективность в лечении коарктации и гипоплазии дуги аорты у детей раннего возраста. Вне зависимости от стратегии хирургической коррекции основное внимание следует уделять индивидуальным анатомическим особенностям дуги аорты, состоянию пациента и сопутствующим врожденным порокам сердца. Однако у всех пациентов необходимо полностью резецировать дуктальную ткань для предотвращения развития рекоарктации аорты и резидуальной артериальной гипертензии.

Ключевые слова: аортопластика; врожденный порок сердца; гипоплазия дуги аорты; детская кардиология; коарктация аорты; легочный гомограф

Introduction

Coarctation of the aorta (CoA) is one of the most common congenital heart defects, accounting for 5–8 % of all congenital heart diseases [1; 2]. This pathology is of particular clinical significance in the neonates due to the high risk of severe complications and mortality in the absence of timely treatment [2]. CoA remains a leading cause of critical conditions in newborns, necessitating early diagnosis and urgent surgical repair [2; 3].

Current clinical guidelines indicate that at least 90 % of patients require surgical intervention during the neonatal period to prevent irreversible target organ damage [4]. In the Russian Federation, a specialized referral system ensures that these patients are transferred to expert cardiac surgery centers.

Despite advancements in care, the choice of the optimal surgical technique remains debated. The primary approaches include resection of the coarctation with an extended end-to-end anastomosis (native tissue repair) and various types of patch aortoplasty [5; 6]. A key unresolved issue is the high incidence of recoarctation (up to 20–30 %) and the persistence of postoperative arterial hypertension [7; 8]. While endovascular techniques are evolving, open surgical intervention remains the standard of care, particularly for neonates [2; 3]. Current guidelines emphasize an individualized approach, considering anatomical features and surgical risks [4].

The aim of this study was to evaluate and compare the early and long-term outcomes of surgical treatment in neonates with coarctation of the aorta using native tissue repair versus patch aortoplasty.

Methods

From 2020 to 2023, a total of 238 patients with coarctation of the aorta and associated congenital heart defects were evaluated at the Departments of Pediatric Cardiac Surgery of the Federal Center for Cardiovascular Surgery (Krasnoyarsk) and the Meshalkin National Medical Research Center (Novosibirsk). The study was approved by the Local Ethics Committee (Protocol No. 2, dated March 24, 2020).

We conducted a two-center prospective randomized study enrolling 105 newborns with coarctation of the aorta and aortic arch hypoplasia who met the inclusion criteria. Randomization was performed using a random number generator.

Sample Size Calculation

The required sample size was calculated using Stata 14 software. Based on literature data regarding recoarctation rates (0 vs. 0.30) [5], it was determined that a minimum of 35 patients per group was sufficient to detect statistically significant differences with adequate power.

Inclusion and Exclusion Criteria

Inclusion criteria: Neonates requiring cardiopulmonary bypass for repair of aortic coarctation and arch hypoplasia.

Exclusion criteria:

- Interrupted aortic arch;
- Critical preoperative condition or shock;
- Active infectious complications (sepsis, pneumonia);
- Severe extracardiac or genetic anomalies incompatible with life;
- Extreme prematurity or birth weight < 1.5 kg;
- Single-ventricle physiology;
- Refusal to participate in the study.

Randomization and Grouping

Patients were randomized into two groups based on the surgical strategy:

Group I (Patch Aortoplasty, $n = 70$): Patients who underwent aortic arch repair using a synthetic or biological patch.

Group II (Native Tissue Repair, $n = 35$): Patients who underwent aortic arch repair using an "aggressive" oblique extended end-to-end anastomosis.

Study Endpoints

Primary endpoint: Development of long-term recoarctation of the aorta (ReCoA), defined by at least one of the following:

Systolic blood pressure gradient > 20 mm Hg between the upper and lower extremities;

Aortic isthmus Z-score < –2 according to computed tomography (CT).

Secondary endpoints:

- Development of arterial hypertension;
- Proportional growth of the aortic arch;
- Compression of the left main bronchus;
- Mortality (in-hospital and long-term).

The study design is presented in Fig. 1.

Diagnostic Criteria and Assessment Methods

The diagnosis of coarctation of the aorta (CoA) was confirmed based on a combination of the following clinical and imaging criteria:

Clinical criteria: A systolic blood pressure gradient > 20 mm Hg between the upper and lower extremities.

Echocardiographic criteria: A peak instantaneous Doppler gradient > 20 mm Hg and/or localized narrowing

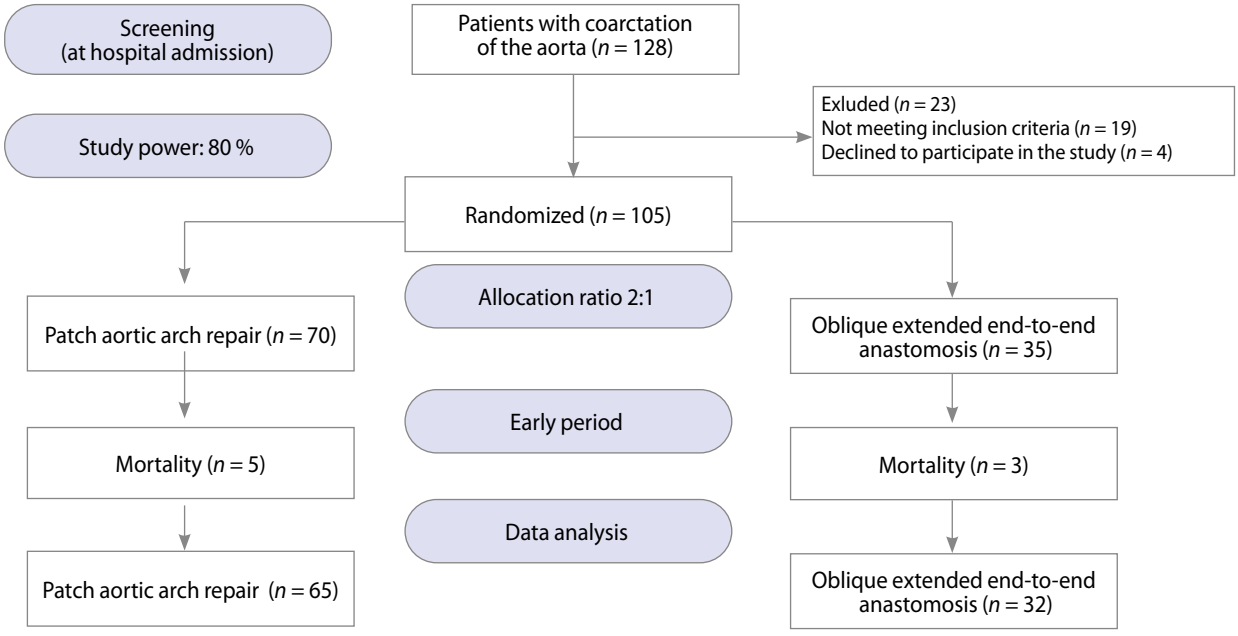

Fig. 1. The study design

of the aortic isthmus relative to the normal diameter (Z-score < –2) [4].

Transthoracic echocardiography was performed according to a standard protocol. Aortic arch hypoplasia was defined as a Z-score < –2 for the proximal and/or distal segments of the arch. Z-scores were calculated using the Parameter(Z) online resource based on reference datasets by Pettersen et al. [9].

Left ventricular (LV) systolic function was assessed using the Teichholz method to determine ejection fraction and fractional shortening. The following parameters were measured: LV end-diastolic diameter (LVEDD), LV end-diastolic volume (LVEDV), interventricular septal thickness (IVSd), and posterior wall thickness (LVPWd).

LV myocardial mass was calculated using the Devereux formula and indexed to body surface area. LV hypertrophy (LVH) was defined as an LV mass index exceeding the 95th percentile for age and sex [9].

Clinical Definitions and Outcomes

In-hospital mortality: Death occurring within 30 days post-surgery or prior to hospital discharge.

Multiple organ failure: Acute dysfunction involving two or more organ systems.

Recurrent laryngeal nerve injury: Vocal cord paresis confirmed by bronchoscopy.

Arterial hypertension: Diagnosed when mean systolic and/or diastolic blood pressure values exceeded the 95th percentile on 24-hour ambulatory blood pressure monitoring (ABPM), confirmed after three independent office blood pressure measurements [8].

Aortic arch aneurysm: Dilation of any aortic segment with a Z-score > +3.

Surgical Technique Preoperative Management and CPB Strategy

Preoperative preparation and intraoperative monitoring followed standard protocols for neonatal cardiac surgery. In all cases, surgical access was achieved via median sternotomy. Operations were performed under cardiopulmonary bypass (CPB) with antegrade cerebral perfusion and moderate hypothermia (28 °C).

The CPB circuit was established using standard cannulation of the aorta and venae cavae. Left ventricular venting was achieved via the right superior pulmonary vein. Following CPB initiation, patients were gradually cooled to the target temperature. Cooling uniformity was monitored using esophageal and rectal temperature probes.

Surgical Repair

Group I (Patch Aortoplasty): The primary procedure involved aortic arch reconstruction. This was performed using either a modified Norwood technique with patch augmentation of the ascending aorta (preserving ductal tissue) or aortic arch repair with ductal tissue excision

Table 1. Baseline clinical and echocardiographic characteristics of the study groups

Variable	Total (*n* = 105)	Patch Aortoplasty (*n* = 70)	Native tissue repair (*n* = 35)	*p*
Age, days	14.00 [8.00; 22.00]	14.00 [8.00; 22.75]	13.00 [7.00; 21.00]	0.749
Height, cm	51.00 [49.00; 54.00]	51.00 [49.00; 53.75]	53.00 [50.00; 54.50]	0.339
Weight, kg	3.10 [2.70; 3.70]	3.00 [2.60; 3.50]	3.30 [3.00; 3.85]	0.054
Body surface area, m^2	0.20 [0.19; 0.23]	0.20 [0.19; 0.23]	0.21 [0.20; 0.24]	0.162
Female sex, *n* (%)	70 (66.7)	46 (65.7)	24 (68.6)	0.942
LV ejection fraction before surgery, %	70.00 [65.00; 75.0]	70.00 [64.25; 75.0]	70.00 [65.00; 74.5]	0.965
Indexed LV end-diastolic volume before surgery, mL/m^2	35.00 [23.90; 43.4]	35.00 [22.05; 40.9]	39.10 [30.75; 44.5]	0.065
Indexed LV end-diastolic diameter before surgery, cm/m^2	7.36 [5.78; 8.4]	7.36 [5.83; 8.4]	7.36 [5.81; 8.5]	0.793
Indexed LV end-systolic diameter before surgery, cm/m^2	3.74 [3.40; 4.4]	3.72 [3.36; 4.2]	3.77 [3.47; 4.4]	0.154
Interventricular septal thickness before surgery, cm	1.84 [1.51; 2.2]	1.86 [1.52; 2.2]	1.81 [1.46; 2.3]	0.731
Posterior LV wall thickness before surgery, cm	2.12 [1.74; 2.5]	2.12 [1.72; 2.5]	2.12 [1.85; 2.4]	0.841
Indexed LV myocardial mass before surgery, g/m^2	40.18 [28.59; 55.1]	40.18 [25.89; 54.3]	40.18 [31.87; 56.6]	0.681
Z-score of ascending aorta before surgery	0.01 [–0.69; 0.8]	0.18 [–0.67; 0.8]	–0.08 [–0.78; 0.6]	0.711
Z-score of proximal aortic arch before surgery	0.85 [–0.04; 1.4]	0.54 [–0.91; 1.4]	1.01 [0.60; 1.5]	0.031
Z-score of distal aortic arch before surgery	–3.02 [–3.53; –2.4]	–3.13 [–3.61; –2.4]	–2.87 [–3.35; –2.6]	0.263
Z-score of aortic isthmus before surgery	–5.93 [–7.28; –4.3]	–6.06 [–7.13; –4.7]	–5.04 [–7.37; –3.95]	0.362

and augmentation using a pulmonary allograft patch (Cardiostar LLC, St. Petersburg, Russia).

Group II (Native Tissue Repair): The primary procedure involved resection of the narrowed aortic segment followed by an extended oblique end-to-end anastomosis, performed without the use of prosthetic material.

Upon completion of the reconstruction, patients were rewarmed to normothermia. The chest was closed in standard fashion, and patients were transferred to the intensive care unit.

Statistical Analysis

Statistical analysis was performed using R software (RStudio environment), Stata 14 (StataCorp LP, College Station, USA), and Python (version 3.x). The normality of distribution for quantitative variables was assessed using the Shapiro – Wilk test. Quantitative variables are presented as median and interquartile range (median [Q1; Q3]), while categorical variables are reported as absolute values and percentages (*n* [%]). Comparisons of quantitative variables between independent groups were performed using the Mann – Whitney U test. Categorical variables were compared using the Pearson χ^2 test or Fisher's exact test, as appropriate.

Table 2. Intraoperative data

Variable	Total (*n* = 105)	Patch Aortoplasty (*n* = 70)	Native tissue repair (*n* = 35)	*p*
Aortic cross-clamp time, min	53.00 [29.00; 64.00]	55.00 [53.00; 68.00]	22.00 [18.50; 31.00]	< 0.001
Intraoperative blood loss, mL	15.00 [11.00; 19.00]	14.00 [11.00; 19.00]	16.00 [12.00; 18.00]	0.804
Intraoperative rectal temperature, °C	36.10 [28.00; 36.40]	28.10 [27.92; 36.10]	36.30 [36.15; 36.50]	< 0.001
Intraoperative urine output, mL	41.00 [31.00; 47.00]	41.50 [31.00; 46.75]	40.00 [29.00; 48.50]	0.688

Table 3. Univariate and multivariate analysis of in-hospital mortality

Variable	Univariate analysis, OR (95 % CI)	*p*	Multivariate analysis, OR (95 % CI)	*p*
Duration of cardiopulmonary bypass	1.035 [1.002–1.070]	0.039	2.240 [1.163–4.314]	0.016
Height	0.405 [0.170–0.964]	0.041	3.281 [0.741–5.210]	0.362
Preoperative inotropic support	4.787 [1.056–21.702]	0.042	7.884 [1.374–45.242]	0.021
Weight	0.385 [0.140–1.061]	0.065	1.021 [0.830–4.832]	0.743
Body surface area	0.436 [0.173–1.102]	0.079	0.843 [0.031–3.955]	0.384
Total anomalous pulmonary venous return	12.867 [0.729–227.075]	0.081	10.323 [0.032–412.377]	0.991

Note. OR – odds ratio; CI – confidence interval.

Table 4. Early postoperative outcomes

Variable	Patch aortic arch repair (*n* = 62)	Native tissue repair (*n* = 31)	*p*
Neurological complications, *n* (%)	9 (14.5)	1 (3.2)	0.156
Renal complications, *n* (%)	5 (8.1)	5 (16.1)	0.292
Paradoxical arterial hypertension, *n* (%)	11 (17.7)	6 (19.4)	0.850
Femoral artery thrombosis, *n* (%)	6 (9.7)	2 (6.5)	0.714
Chylothorax, *n* (%)	2 (3.2)	0 (0.0)	0.551
Left bronchial compression, *n* (%)	9 (14.5)	11 (35.5)	0.020
Recurrent laryngeal nerve injury, *n* (%)	5 (8.1)	4 (12.9)	0.475
Phrenic nerve injury, *n* (%)	3 (4.8)	0 (0.0)	0.548
Peak gradient across the aortic isthmus, mmHg	13 [11; 16.5]	9 [7; 12.5]	0.002
LV ejection fraction, %	66.66 [63.42; 70.61]	65.31 [60.34; 69.37]	0.191
Indexed LV end-diastolic volume, mL/m^2	44.06 [37.74; 57.35]	45.51 [30.62; 56.79]	0.675
Indexed LV end-diastolic diameter, mm/m^2	9.28 [7.59; 10.56]	8.95 [6.64; 10.69]	0.634
Indexed LV end-systolic diameter, mm/m^2	4.68 [4.19; 5.29]	5.07 [4.33; 5.57]	0.139
Interventricular septal thickness, mm	2.38 [1.88; 2.77]	2.18 [1.79; 2.82]	0.436
Posterior LV wall thickness, mm	2.60 [2.15; 3.17]	2.78 [2.13; 3.14]	0.971
Indexed LV myocardial mass, g/m^2	49.43 [31.75; 66.43]	50.01 [35.37; 72.26]	0.538

Note. iLVEDV – indexed left ventricular end-diastolic volume; iLVEDD – indexed left ventricular end-diastolic diameter; iLVESD – indexed left ventricular end-systolic diameter; LV – left ventricle; EF – ejection fraction. Data are presented as median (interquartile range) or *n* (%).

Predictors of early and late postoperative complications were identified using univariate and multivariable logistic regression models. Survival analysis regarding freedom from recoarctation and arterial hypertension was conducted using the Kaplan – Meier method. Time-to-event analysis for adverse outcomes was performed using Cox proportional hazards regression to determine associations with the studied variables. A p-value < 0.05 was considered statistically significant.

Results

The basic characteristics of the groups are presented in Table 1.

As shown in Table 1, the only difference in baseline characteristics between the study groups was the Z-score of the proximal aortic arch before surgery, which was significantly higher in the native tissue repair group.

Intraoperative data are presented in Table 2.

As shown in Table 2, the Native Tissue Repair group demonstrated significantly shorter aortic cross-clamp times compared with the Patch Repair group. Conversely, the Patch Repair group had significantly lower minimum rectal temperatures during CPB.

In-hospital mortality did not differ significantly between groups: 5 patients (7.1 %) in the Patch Repair group versus 3 patients (8.6 %) in the Native Tissue Repair group (p > 0.999). Predictors of in-hospital mortality based on uni- and multivariable analyses are presented in Table 3.

Early postoperative outcomes and complications are presented in Table 4.

Early postoperative outcomes (Table 4) revealed that the peak instantaneous Doppler gradient across the aortic isthmus was significantly higher in the Patch Repair group. However, compression of the left main bronchus was more frequently observed in the Native Tissue Repair group.

Long-term outcomes

The median follow-up period was 34 months (IQR: 27–40 months), with a follow-up completeness of 96.8 %. Long-term outcomes are presented in Table 5.

As shown in Table 5, significant differences between the groups were observed for arterial hypertension and peak gradient across the aortic isthmus, which were significantly lower in the native tissue repair group, whereas the Z-score of the aortic isthmus was significantly lower in the patch repair group.

Figure 2 shows two Kaplan – Meier curves corresponding to the groups of patients who underwent patch aortic arch repair and native tissue aortic arch repair. The vertical axis represents the proportion of patients free from the event (arterial hypertension), while the

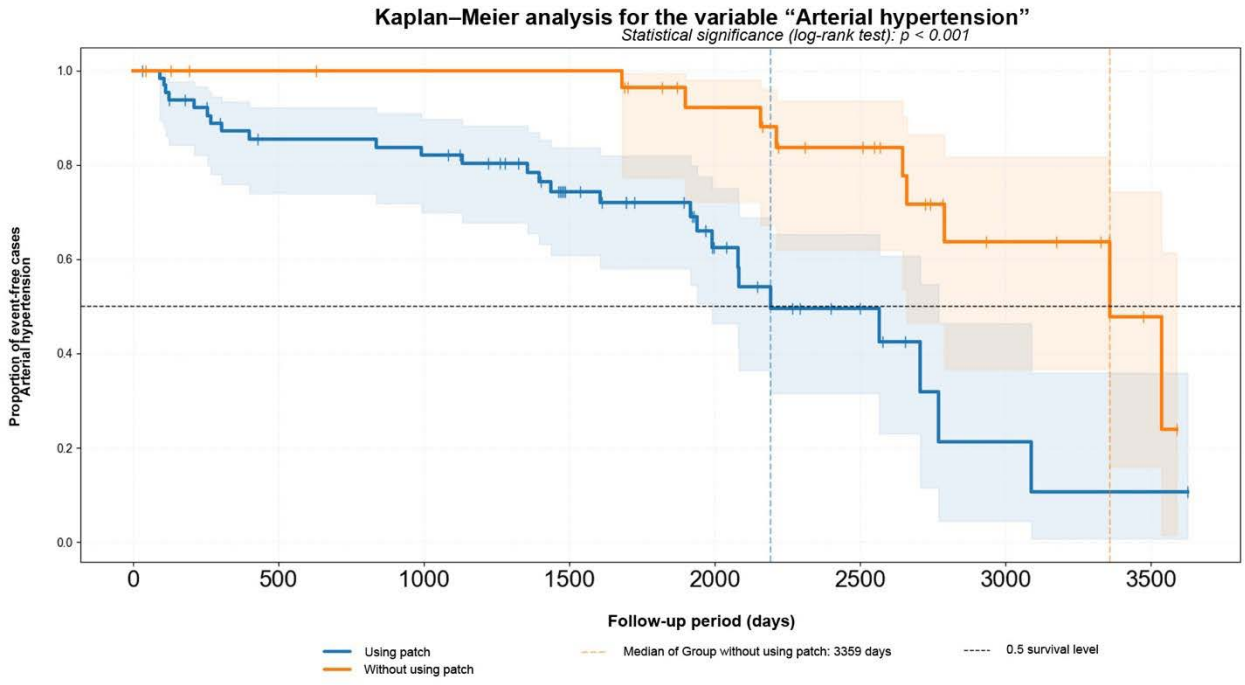

Fig. 2. Kaplan – Meier curves for arterial hypertension

Table 5. Long-term postoperative outcomes and complications

Variable	Total (*n* = 90)	Patch aortic arch repair (*n* = 59)	Native tissue repair (*n* = 31)	*p*
Arterial hypertension, *n* (%)	36 (40.0)	28 (47.5)	8 (25.8)	0.046
Recoarctation of the aorta requiring reoperation, *n* (%)	21 (23.3)	17 (28.8)	4 (12.9)	0.090
Subclavian artery stenosis, *n* (%)	1 (1.1)	1 (1.7)	0 (0.0)	> 0.999
Descending aortic aneurysm, *n* (%)	1 (1.1)	1 (1.7)	0 (0.0)	> 0.999
Carotid or brachiocephalic artery stenosis, *n* (%)	1 (1.1)	1 (1.7)	0 (0.0)	> 0.999
Growth rate of proximal aortic arch, μm/day	5.95 [4.39; 7.39]	6.19 [5.24; 8.59]	5.79 [4.05; 7.15]	0.121
Growth rate of distal aortic arch, μm/day	7.21 [5.94; 9.15]	6.82 [6.31; 8.29]	7.22 [5.67; 9.22]	0.504
Rate of anastomosis Z-score increase, $\times 10^{-3}$/day	33.72 [26.27; 41.12]	33.31 [25.41; 40.14]	34.10 [28.47; 41.72]	0.214
LV ejection fraction, %	67.46 [64.03; 72.66]	69.29 [64.78; 73.08]	66.45 [61.32; 70.45]	0.063
Indexed LV end-diastolic volume, mL/m^2	45.78 [34.10; 57.02]	46.15 [35.05; 55.82]	44.10 [32.25; 57.23]	0.492
Indexed LV end-diastolic diameter, mm/m^2	11.06 [8.48; 12.63]	10.92 [8.89; 12.62]	11.07 [7.93; 12.53]	0.786
Indexed LV end-systolic diameter, mm/m^2	5.75 [5.16; 6.45]	5.66 [5.11; 6.54]	5.85 [5.31; 6.41]	0.382
Interventricular septal thickness, mm	2.76 [2.18; 3.36]	2.84 [2.18; 3.44]	2.58 [2.12; 3.29]	0.262
Posterior LV wall thickness, mm	3.05 [2.49; 3.73]	2.95 [2.50; 3.72]	3.41 [2.50; 3.78]	0.747
Indexed LV myocardial mass, g/m^2	58.78 [37.86; 83.16]	58.70 [36.80; 82.99]	59.05 [43.18; 83.10]	0.535
Peak gradient across the aortic isthmus, mmHg	21 [18; 24]	24 [19; 27]	18 [17; 22]	0.001
Z-score of proximal aortic arch	0.80 [0.13; 1.16]	0.82 [0.15; 1.20]	0.75 [0.10; 1.12]	0.165
Z-score of distal aortic arch	0.64 [0.07; 1.45]	0.70 [0.10; 1.50]	0.61 [0.00; 1.37]	0.091
Z-score of aortic isthmus	0.26 [0.00; 1.09]	0.10 [–0.20; 0.80]	0.43 [0.15; 1.30]	0.048

Note. BCA – brachiocephalic artery; iLVEDV – indexed left ventricular end-diastolic volume; iLVEDD – indexed left ventricular end-diastolic diameter; iLVESD – indexed left ventricular end-systolic diameter; LV – left ventricle; SA – subclavian artery; EF – ejection fraction. Data are presented as median (interquartile range) or *n* (%).

horizontal axis indicates the duration of follow-up in days. The log-rank test value was $p < 0.001$, indicating statistically significant differences between the study groups.

The only independent predictor of arterial hypertension was the peak gradient across the aortic isthmus before discharge, which increased the risk by 4.7-fold for each 1 mmHg increase.

Figure 3 shows two Kaplan – Meier curves corresponding to the groups of patients who underwent patch repair and native tissue aortic arch repair. The log-rank test value

Table 6. Cox proportional hazards regression analysis for arterial hypertension

Variable	Univariate analysis			Multivariate analysis		
	Coefficient	Hazard ratio (95 % CI)	*p*	Coefficient	Hazard ratio (95 % CI)	*p*
Peak gradient across the aortic isthmus before discharge, mmHg	1.54	4.68 [2.69; 8.14]	<0.001	1.49	4.42 [2.50; 7.84]	<0.001
Patch material (homograft patch)	0.93	2.53 [1.30; 4.92]	0.006	0.68	1.98 [0.89; 4.41]	0.096
Preoperative mitral valve Z-score	–0.54	0.58 [0.37; 0.91]	0.017	–0.18	0.83 [0.49; 1.41]	0.498
Preoperative aortic valve Z-score	–0.63	0.53 [0.31; 0.92]	0.025	–0.32	0.73 [0.41; 1.30]	0.284
Age, days	–0.49	0.61 [0.37; 1.02]	0.061	–	–	–
Peak gradient across the aortic isthmus after surgery, mmHg	–0.56	0.57 [0.30; 1.07]	0.082	–	–	–
Indexed LV end-diastolic volume before surgery, mL/m²	–0.37	0.69 [0.43; 1.10]	0.117	–	–	–
Preoperative Z-score of distal aortic arch	–0.39	0.68 [0.42; 1.10]	0.119	–	–	–
Postoperative LV ejection fraction, %	0.38	1.46 [0.88; 2.44]	0.148	–	–	–

was $p = 0.147$, indicating the absence of statistically significant differences between the study groups.

Cox regression analysis for recoarctation of the aorta is presented in Table 7.

As shown in Table 7, significant risk factors for the development of recoarctation of the aorta were the peak gradient across the aortic isthmus before discharge, which increased the risk of recoarctation by 3.44-fold,

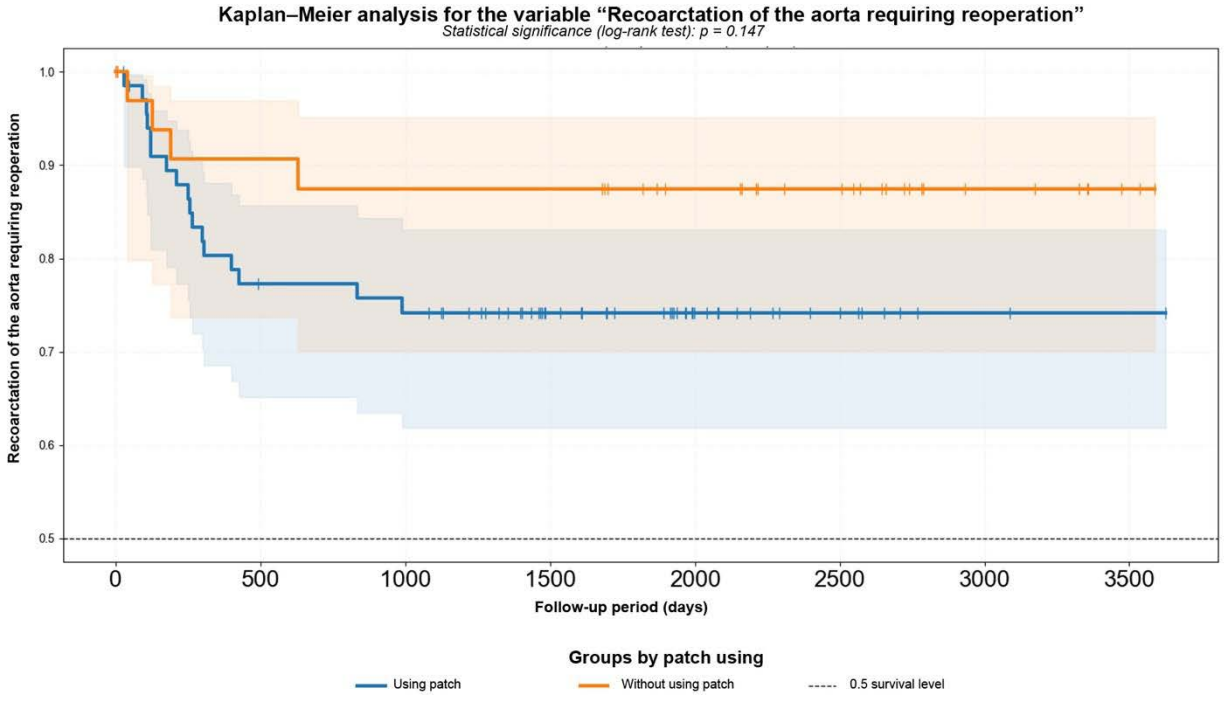

Fig. 3. Kaplan – Meier intergroup analysis of recoarctation of the aorta requiring reoperation

Table 7. Cox proportional hazards regression analysis for recoarctation of the aorta

Variable	Univariate analysis			Multivariate analysis		
	Coefficient	Hazard ratio (95 % CI)	*p*	Coefficient	Hazard ratio (95 % CI)	*p*
Peak gradient across the aortic isthmus before discharge, mmHg	1.24	3.44 [1.84; 6.42]	<0.001	1.13	3.11 [1.59; 6.08]	0.001
Excision of ductal tissue	–1.32	0.27 [0.11; 0.65]	0.003	–0.80	0.45 [0.16; 0.75]	0.012
Preoperative Z-score of proximal aortic arch	–0.41	0.66 [0.47; 0.94]	0.021	–0.26	0.77 [0.52; 1.14]	0.193
Preoperative LV ejection fraction, %	0.67	1.96 [1.00; 3.81]	0.048	0.73	2.08 [0.98; 4.41]	0.057
Preoperative aortic valve Z-score	–0.50	0.60 [0.33; 1.12]	0.110	–	–	–
Preoperative LV myocardial mass index, g/m^2	–0.61	0.55 [0.25; 1.18]	0.125	–	–	–

and excision of ductal tissue, which reduced the risk of recoarctation by 3.7-fold.

Figure 4 presents a Kaplan – Meier analysis of time to development of recoarctation of the aorta according to the peak gradient across the aortic isthmus before discharge. The sample was divided into two groups based on the optimal cutoff value (≤ 14 mmHg and > 14 mmHg), determined using a statistical criterion.

Kaplan – Meier analysis demonstrated that patients with a peak gradient ≤ 14.0 mmHg (blue curve) had a significantly higher proportion of event-free survival (recoarctation of the aorta requiring reoperation) throughout the entire follow-up period compared with patients

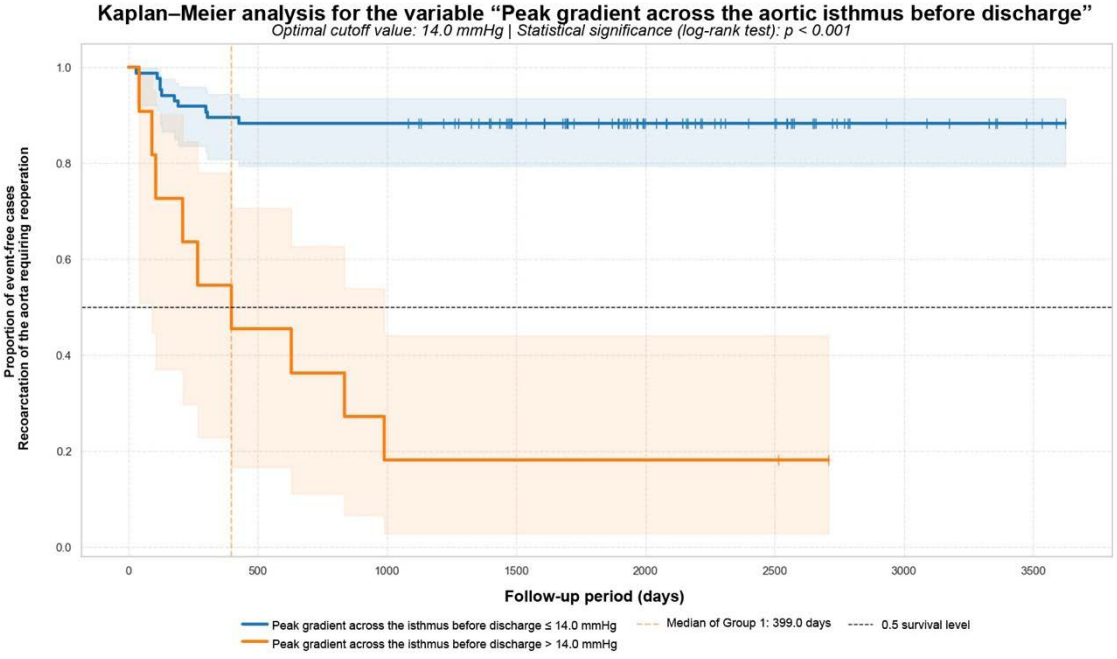

Fig. 4. Kaplan – Meier intergroup analysis of recoarctation of the aorta requiring reoperation according to the peak gradient across the aortic isthmus before discharge

whose peak gradient exceeded 14.0 mmHg (orange curve).

Discussion

The issue of aortic coarctation in neonates remains highly relevant in pediatric cardiac surgery [9]. Despite significant progress in surgical treatment, this pathology continues to be a cause of serious complications and high mortality rates in early infancy [10]. Extended end-to-end anastomosis remains the "gold standard" for treating aortic coarctation in children; however, many centers employ patch aortoplasty to effectively widen the aortic arch [8–12].

At the same time, there is no consensus within the scientific community regarding the optimal surgical technique for CoA correction. This has necessitated numerous studies aimed at comparing the outcomes of various surgical correction methods to determine the most effective treatment strategy for this condition [12–24].

The peak pressure gradient across the aortic isthmus serves as a key indicator reflecting the efficacy of surgical treatment. Surgical repair leads to a significant reduction in this gradient, which subsequently decreases blood pressure in the upper body and reduces afterload on the left ventricle [6]. In the postoperative period, a residual pressure gradient following aortic arch repair is not uncommon and may cause severe complications both in the early postoperative period (e.g., acute heart failure, acute kidney injury, necrotizing enterocolitis) and in the long term (arterial hypertension, aortic aneurysms, ischemic heart disease, cerebrovascular aneurysms) [1; 6; 14]. A number of studies have demonstrated that surgical elimination of the pressure gradient across the aortic arch, regardless of patient age, is associated with favorable long-term outcomes, a low complication rate, and reduced late mortality [15–18]. Our study established that a reduction in the pressure gradient was accompanied by significant improvement in clinical status in the early postoperative period. Moreover, a comparison of the two surgical techniques revealed no significant differences between them. Similar conclusions were reached in other works, which also found no statistically significant differences between the results of surgical methods for aortic coarctation correction [12; 17]. This suggests that the decisive factor influencing pressure gradient reduction is the successful surgical intervention itself, rather than the specific technical modification of the operation.

Hospital mortality following surgical correction of aortic coarctation occurs in approximately 10 % of cases [5]. According to the literature, the main risk factors for mortality in the early postoperative period are patient comorbidities, low birth weight and prematurity, progressive heart failure in neonates, acute kidney injury, various infectious diseases (including congenital pneumonia, enterocolitis), and severe neurological symptoms [5; 7; 25]. Additionally, continuous infusion of prostaglandin E_1 is administered to neonates in the preoperative period to maintain patency of the ductus arteriosus. Prolonged prostaglandin infusions can lead to the development of enterocolitis, complicated by sepsis [26; 27]. According to a study by Soynov I.A. et al., no hospital mortality was noted when analyzing the use of the "extended end-to-end anastomosis" surgical correction method and patch aortic arch angioplasty [5]. In our study, hospital mortality showed no significant differences between the groups and occurred with similar frequency (8.6 % vs. 7.1 %, $p = 0.999$). The only risk factor for mortality, regardless of the surgical technique used, was the duration of cardiopulmonary bypass (95 % CI [1.374; 45.242], $p = 0.016$).

In the early postoperative period following aortic coarctation repair, bronchial compression by the aortic arch occurs in 0.7–2.2 % of cases, leading to acute respiratory failure [22]. In a study by Hui C. et al., compression of the left main bronchus occurred in 22 % of patients undergoing extended oblique anastomosis arch repair, and in 12 % utilizing a patch [22]. Our study results were comparable; bronchial compression was significantly more frequent in the group undergoing extended end-to-end anastomosis (35.5 % vs. 14.5 %, $p = 0.020$) in the early postoperative period.

The development of aortic recoarctation in the late postoperative period following primary surgical correction is a significant complication, with rates reaching 44 % in various studies [23]. A hemodynamic marker confirming significant obstruction is a pressure gradient between the proximal and distal aortic arch sections exceeding 20 mmHg [24]. In our study, the sole predictor for the development of late aortic recoarctation provided was a peak pressure gradient at the isthmus exceeding 14 mmHg (HR = 3.75; $p < 0.001$). Complete removal of ductal tissue acted as a significant predictor (HR = 0.33; $p = 0.017$), reducing the risk of recoarctation by preventing fibrosis ($p < 0.001$).

The obtained data align with findings from previous studies and confirm that surgical treatment effectively reduces the peak pressure gradient at the aortic isthmus. The absence of statistically significant differences between the compared groups underscores the need for further research to identify factors influencing long-term outcomes. Analysis of results at 6 and 12 months, as well as during delayed follow-up, suggests that differences in the efficacy of surgical correction methods for neonatal

aortic coarctation may manifest over a longer observation period, such as 5 years.

Summarizing all obtained results, including the analysis of confounding factors, it can be concluded that the outcomes of operations performed with and without a patch did not differ statistically significantly. However, the complete removal of ductal tissue was of critical importance.

Thus, both common surgical strategies demonstrate comparable efficacy in treating coarctation and aortic arch hypoplasia in neonates. Regardless of the surgical correction strategy, primary attention should be paid to the individual anatomical features of the aortic arch, patient status, and concomitant congenital heart defects. However, in all patients, it is imperative to completely resect all ductal tissue to prevent the development of aortic recoarctation and residual arterial hypertension.

Study Limitations

Our study has several limitations that should be considered when interpreting the results. First, despite the prospective randomized design, the sample size was relatively small (105 patients), which may have limited the statistical power regarding rare postoperative complications. Second, the median follow-up period was 34 months, reflecting early and mid-term rather than long-term outcomes. A longer monitoring period (10 years or more) is required to definitively assess the risks of aneurysm formation at the reconstruction site or the development of resistant arterial hypertension in adulthood. Third, the study was conducted across two cardiac surgery centers. Although surgical protocols and selection criteria were standardized, the influence of individual surgeon experience and subtle differences in perioperative management strategies cannot be entirely ruled out. Finally, the exclusion criteria (body weight less than 1.5 kg, critical preoperative condition, sepsis) limit the generalizability of our findings to the cohort of the most critically ill patients and preterm infants with extremely low birth weight.

Conclusions

The primary predictor of late recoarctation was a discharge peak trans-isthmic gradient > 14 mm Hg (HR = 3.75; $p < 0.001$). Conversely, complete excision of ductal tissue acted as a significant protective factor (HR = 0.33; $p = 0.017$), reducing the risk of recurrence.

In-hospital mortality was comparable between the native tissue and patch repair groups (8.6 % vs. 7.1 %; $p = 0.999$). The only independent risk factor for mortality was the duration of cardiopulmonary bypass (95 % CI [1.37; 45.24]; $p = 0.016$).

Compression of the left main bronchus in the early postoperative period occurred significantly more frequently in the extended oblique end-to-end anastomosis group compared to the patch repair group (35.5 % vs. 14.5 %; $p = 0.020$).

Late arterial hypertension was a common complication (23.7 %). The absence of complete ductal tissue excision significantly increased the risk of hypertension (OR = 3.84; $p = 0.013$). Patch aortoplasty was associated with a reduced risk of arterial hypertension (OR = 0.39; $p = 0.046$). Although native tissue repair showed a trend toward lower recoarctation rates (12.9 % vs. 25.4 %), this difference did not reach statistical significance ($p = 0.266$).

Proportional growth of the anastomosis appeared independent of the specific surgical technique. However, complete excision of ductal tissue was essential for promoting proportional growth and minimizing early restenosis.

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