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Risk factors of mortality and recoarctation after coarctation repair in infancy

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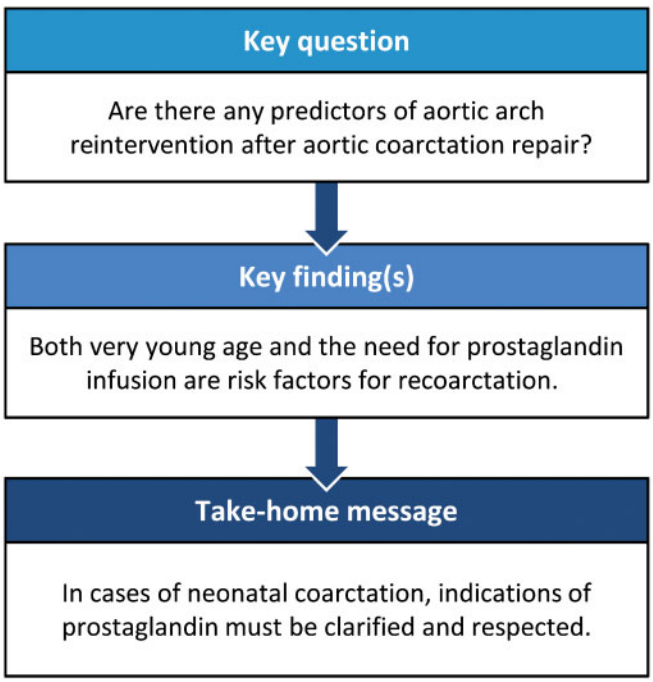

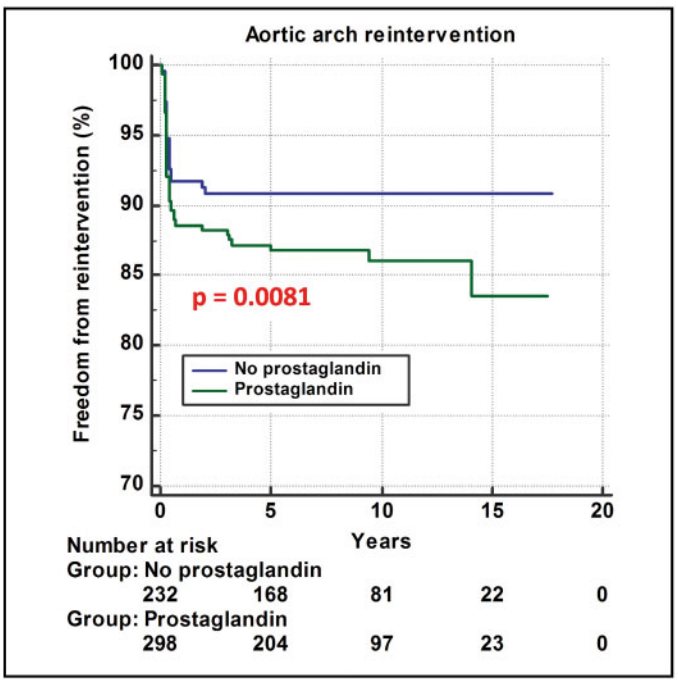

Abstract

OBJECTIVES: The main challenge of aortic coarctation (CoA) repair in infants is to obtain durable results without morbidity. We aimed to describe predictors of aortic arch reintervention after aortic CoA repair.

METHODS: Between January 2000 and March 2014, we retrospectively included consecutive infants with isolated CoA or CoA with ventricular septal defect (CoA + VSD) who had surgical repair of the aortic arch before 3 months of age.

RESULTS: Five hundred and thirty patients were included: 308 (58%) patients had isolated CoA and 222 (42%) patients had CoA + VSD. Three hundred and eighty-five patients (72.6%) had CoA repair, 51 patients (9.6%) had CoA repair with closure of VSD and 94 patients

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(17.8%) had CoA repair with pulmonary artery banding. Mean age at operation was 13 ± 1.6 days, with 294 patients (55.5%) operated on before 2 weeks. Median follow-up was 7.57 years. Sixty-one patients (11.5%) needed reintervention on the aortic arch. Freedom from aortic arch reintervention was 90% at 1 year and 88.5% at 5 years. Proportions of aortic arch reintervention were similar in the different surgical strategy groups ($P = 0.80$). However, in patients receiving prostaglandin E1 (PGE1), the end-to-end repair was at higher risk of recoarctation compared to the extended end-to-side repair ($P = 0.033$). The risk factors of aortic arch reintervention were age at repair <15 days ($P = 0.034$) and the need for PGE1 infusion at surgery ($P = 0.0043$).

CONCLUSIONS: CoA repair in young infants has an overall good outcome. The use of PGE1 may modify the aortic arch anatomy and mask the boundaries of the resection to be performed. PGE1 treatment should be studied more specifically in another study to improve preoperative management.

Keywords: Paediatrics • Coarctation • Congenital heart disease • Surgery • Outcome

INTRODUCTION

The main challenge of repair of aortic coarctation (CoA) is to obtain durable reparation without late morbidity. The clinical condition at time of diagnosis or the associated cardiac defects can complicate the surgical repair of the aortic arch. Prenatal diagnosis has suspected CoA of the aorta in less than half of the cases in our area [1]. Prostaglandin E1 (PGE1) is used in some neonates to try to reopen the arterial duct or the aortic isthmus to avoid clinical aggravation while awaiting surgery. This treatment may enable to wait a few days to perform the aortic arch repair, but it does also include its own risks and adverse effects [2, 3].

Despite multiple studies focusing on the impact of the technique of repair on mortality and proportion of recoarctation, a limited number of risk factors for recoarctation have been identified [4, 5]. Child clinical status, particularly weight at surgery, anatomical characteristics of the aortic arch or surgical strategy have been proposed as risks factors for adverse outcome, including recurrent obstruction [6–10]. Early repair is the commonly chosen strategy to correct CoA with a large proportion of patients being operated on within the first months of life. Here, we sought to analyse the outcomes after CoA surgical repair in infants under 3 months of age to identify risk factors for mortality and aortic arch reoperation.

PATIENTS AND METHODS

Study population

We reviewed all consecutive patients who underwent CoA repair before 3 months of age at our institution between January 2000 and March 2014.

We included all patients with an isolated CoA or with ventricular septal defect (CoA + VSD). Patients with other congenital heart diseases were excluded.

Demographic data included gender, prenatal or postnatal diagnosis, term, associated genetic anomalies and comorbidities.

Anatomical description of the aortic arch was analysed from echocardiographic records. The presence of a left superior caval vein, size of the mitral valve, aortic valve anomalies, presence of hypoplastic transverse aorta (diameter < 3 mm), patency of the arterial duct, size of the left ventricle, number and anatomical characteristics of VSD were noted. The need for PGE1 infusion was also noted. In our cohort, the PGE1 indications were: heart failure, lower limbs systemic perfusion from the arterial duct related to aortic arch hypoplasia, severe pulmonary hypertension, systemic hypertension and hypoplasia of the aortic arch to prevent left heart failure.

In addition, in our centre, a percutaneous treatment of neonatal aortic CoA presenting with severe left ventricular dysfunction as a bridge to surgery was performed [11]. This strategy was also noted.

Surgical technique

Surgical data included age and weight at time of repair, surgical strategy, aortic cross-clamping time, and if an extracorporeal circulation was performed.

Three surgical strategies were used: (i) CoA repair (CoA repair), (ii) CoA repair and VSD closure (CoA + VSD closure) or (iii) CoA repair with pulmonary artery banding (CoA + PAB). Four specific techniques for CoA repair were used: Crafoord (end-to-end), modified Crafoord (most extensive possible resection with release of 2 sides of the aortic arch to the ascending aorta and to the arterial trunk innominate, the incision is oblique with a long slit; see Supplementary material, Video S1), patch augmentation of the arch and subclavian flap angioplasty. Section of the left subclavian artery (if the resection of the subclavian artery was needed to resect the CoA area and then perform the anastomosis) was also reported. Surgery was mostly performed through left thoracotomy, except for anatomical difficulties (i.e. thoracic malformation or certain hypoplastic transverse aorta) or surgery with extracorporeal circulation.

After surgery, duration of mechanical ventilation, length of stay in intensive care unit and hospitalization, and reinterventions on aortic arch (delay between repair and the first reoperation, the type and the number of reintervention) were recorded. The reintervention indications were the same criteria as those of the initial operative indication (diagnostic criteria of CoA). We also collected data on other reoperations (secondary VSD closure, pulmonary de-banding, reoperation on intracardiac and aortic valve obstructive lesions).

At the last follow-up, arterial pressure, pressure gradient between upper and lower limbs, cardiac treatment and echocardiographic data detailing the aortic arch anatomy were noted.

This study had been approved by our ethics board (Comité de Protection des Personnes—Ile-de-France VI).

Statistical analysis

Continuous variables are presented as the mean value ± standard deviation. Categorical variables were presented as frequencies

Table 1: Patient characteristics

Variables	
Demographic characteristics, *n* (%)	
Male/female	311 (58.7)/219 (41.3)
Prenatal diagnosis/postnatal diagnosis	231 (43.6)/299 (56.4)
Prematurity/full term	67 (12.6)/463 (87.4)
Genetic abnormalities	21 (3.9)
Turner's syndrome	7/21 (33)
Comorbidities	
Polymalformation syndrome	17 (3.2)
Intrauterine growth retardation	13 (2.4)
Postnatal diagnosis, *n* (%)	299 (56.4)
Median age at diagnosis (days) (1st–3rd quartile)	9 (0–80)
Days (0–3), *n* (%)	61 (20.4)
Days (4–10), *n* (%)	110 (36.8)
Days (11–29), *n* (%)	82 (27.4)
Days >29, *n* (%)	46 (15.4)
Clinical symptoms for postnatal diagnosis, *n* (%)	
Pulse abolition (alone/association)	38 (12.7)/134 (44.8)
Heart murmur (alone/association)	26 (8.7)/80 (26.7)
High blood pressure (alone/association)	5 (1.7)/44 (14.7)
Heart failure (alone/association)	19 (6.3)/86 (28.8)
Cardiogenic shock (alone/association)	38 (12.7)/75 (25.1)
Other	12 (4)
Anatomical characteristics	
Persistent left superior caval vein, *n* (%)	63/530 (11.9)
Mitral valve	
Mitral size	
Median size (mm) (1st–3rd quartile)	9 (8.5–10)
Normal size	316
<-2DS	34
Unknown	25
VSD, *n* (%)	222/530 (41.9)
Number of VSD, *n* (%)	
One	185/222 (83.3)
Perimembranous	96/222 (43.2)
Muscular	73/222 (32.9)
Outlet	26/222 (11.7)
Inlet	1/222 (0.5)
Multiple	37/222 (16.7)
Restrictive VSD	80/222 (36)
Aortic valve, *n* (%)	
Bicuspid	275 (51.9)
Tricuspid	253 (47.7)
Monocusp	2 (0.4)
Transverse aorta, *n* (%)	
<3 mm (hypoplasia)	161 (30.4)
≥3 mm	369 (69.6)

VSD: ventricular septal defect.

and percentages. Comparisons of categorical variables were made using the Fisher's exact test (cell size ≤ 5) or χ^2 test when appropriate (cell size > 5). Time to death and time to reoperation are shown in Kaplan–Meier curves and all survival curves start at the time of surgery. Cox proportional hazard regression analysis, with the date of repair used as start date, was performed as univariable and multivariable analysis to investigate risk factors for death and reoperation. When the number of events was under 10 outcome events per predictor variable, we used the χ^2 test or Mann–Whitney *U*-test to determine it as appropriate. All entered variables were selected based on clinical experience, univariable analysis and previously published data. For all analyses, a 2-tailed *P*-value <0.05 was used as the criterion for statistical significance. Analysis was conducted using *Medcalc* (MedCalc Software, Mariakerke, Belgium).

RESULTS

Population description

We included 530 patients: 308 isolated CoA (58.1%) and 222 CoA + VSD (41.9%). Patient characteristics are summarized in Table 1.

Nineteen (3.6%) patients had previous dilatation of CoA for severe cardiogenic shock at the median age of 11 ± 4.8 days.

The mean age at surgery was 13 ± 1.6 days. Mean weight at repair was 3.2 ± 0.75 kg, with 68 patients (12.8%) weighing <2.5 kg.

Three hundred and eighty-five (73%) patients underwent CoA repair, 51 (10%) patients had CoA repair with concomitant VSD closure and 94 (17%) patients had CoA repair with PAB. The PAB was a classical banding for 28 (30%) patients, 35 (37%) patients had absorbable banding and 30 (32%) patients dilatable banding.

Surgery was performed through thoracotomy in 461 patients [CoA repair (*n* = 368) and CoA + PAB (*n* = 93)] and through sternotomy in 69 patients [CoA repair (*n* = 17), CoA + VSD closure (*n* = 51) and CoA + PAB (*n* = 1)].

Types of aortic arch repair were 406 (77%) modified Crafoord (extended end-to-side), 88 (16%) Crafoord (end-to-end), 31 (5.8%) aortoplasties with patch and 5 (1%) subclavian flaps. The subclavian artery needed to be sacrificed in 156 patients (29%).

There were 69 (13%) surgeries using extracorporeal circulation, the mean aortic clamping time was 28 ± 22.6 min, while it was 17 ± 22.3 min for surgery without extracorporeal circulation (*P* = 0.06).

The median length of intubation was 1 (range 1–3) day, length of stay in intensive care unit 4 (3–7) days and duration of hospitalization 12 (9–17) days.

Median follow-up was 7.57 (range 0.25–15.98) years.

At the last follow-up, 35 patients (6.6%) were symptomatic. Main symptom was dyspnoea (*n* = 31) at the last evaluation. Thirty-seven patients had a pressure gradient >20 mmHg and 54 patients received a treatment for heart failure.

Aortic arch reintervention

Sixty-one patients (11.5%) needed reintervention for recurrent or residual aortic arch obstruction. The types and the number of reinterventions (surgery and endovascular interventions) are described in Table 2. There was no statistical difference among the 3 surgical strategies (*P* = 0.80) (Fig. 1).

Delay before reintervention on the aortic arch was 112 days (95% confidence interval 46–1131), 113 days (74–1326) and 114 days (65–1115), respectively, for CoA alone, CoA + VSD and CoA + PAB (*P* = 0.89).

In univariable analysis, risk factors of reintervention are summarized in Table 3. In multivariable analysis, the age at repair <15 days (*P* = 0.032), and the need for PGE1 infusion (*P* = 0.0072) were significantly associated with recoarctation (Fig. 2).

Prostaglandin E1

Two hundred and ninety-eight patients (56.2%) received PGE1 before surgery. The indications were heart failure (*n* = 163, 54.7%), lower limbs systemic perfusion from the arterial duct related to aortic arch hypoplasia (*n* = 23, 7.7%), severe pulmonary hypertension (*n* = 20, 6.7%), systemic hypertension (*n* = 17, 5.7%), hypoplasia of the aortic arch to prevent left heart failure (*n* = 66,

Table 2: Aortic arch reintervention and type of initial surgery

	CoA alone (*n* = 385)	CoA + VSD closure (*n* = 51)	CoA + PAB (*n* = 94)	Total (*n* = 530)
Reintervention (*n*=61), *n* (%)	43 (11.2)	7 (13.7)	11 (11.7)	61 (11.5)
Surgery (*n*=3)	2 (0.5)	0	1 (1.1)	3 (0.6)
CoA repair	2 (0.5)	0	1 (1.1)	3 (0.6)
Endovascular (*n*=58)	41 (10.6)	7 (13.7)	10 (10.6)	58 (11.1)
Dilatation	39 (10.1)	7 (13.7)	10 (10.6)	56 (10.7)
Dilatation + stent	2 (0.5)	0	0	2 (0.4)
Number of reinterventions				
1 time	35	4	5	44
2 times	7	1	5	13
3 times	1	1	1	3
>3 times	0	1	0	1

CoA: coarctation; PAB: pulmonary artery banding; VSD: ventricular septal defect.

Table 3: Univariable analysis of risk factors for aortic arch reintervention

Covariates	Exp(*b*)	95% CI of Exp(*b*)	*P*-value
Demographic variables			
Prematurity	1.23	0.60–2.49	0.57
Postnatal diagnosis	0.90	0.54–1.51	0.70
Associated anomalies and morphometric variables			
Left superior caval vein	0.83	0.36–1.94	0.67
Mitral valve size <-2SD	0.71	0.31–1.65	0.43
VSD	0.93	0.55–1.55	0.77
Biscuspid aortic valve	0.73	0.44–1.21	0.22
Hypoplastic transverse aorta (<3 mm)	0.91	0.52–1.59	0.74
Clinical variables			
Arterial duct open before surgery	1.02	0.58–1.77	0.95
Dilatation in emergency before surgery	1.62	0.51–5.17	0.42
PGE1 infusion at surgery	1.53	0.90–2.60	**0.0043**
Surgical variables			
Weight at surgery <2.5 kg	0.77	0.53–1.11	0.16
Age at surgery <15 days	0.98	0.97–1.00	**0.034**
Sternotomy	1.05	0.50–2.20	0.90
Type of surgical strategy	1.04	0.76–1.43	0.80
Type of aortic arch repair	0.80	0.48–1.33	0.39
Left subclavian conservation	0.74	0.44–1.25	0.26
Extracorporal circulation	0.88	0.40–1.93	0.75

CI: confidence interval; PGE1: prostaglandin E1; VSD: ventricular septal defect.

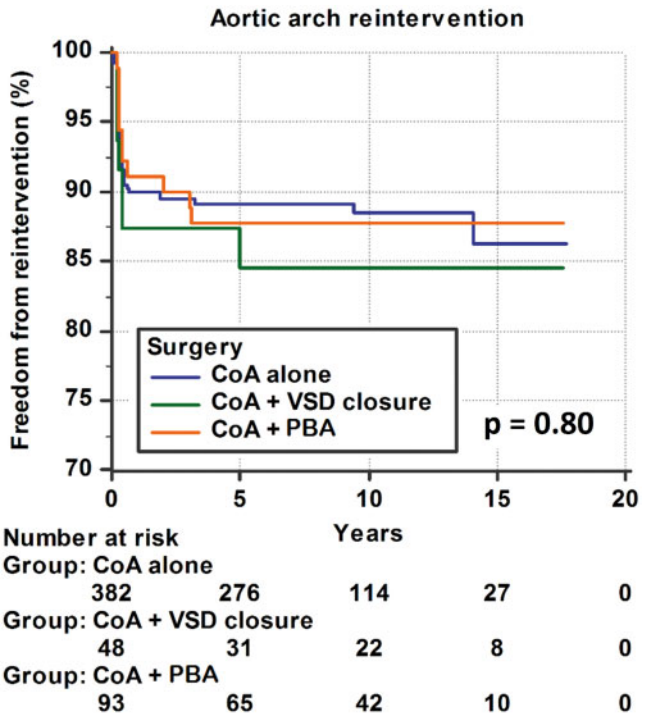

Figure 1: Aortic arch reintervention (surgery and endovascular interventions) by surgical strategy.

22.2%) and precise indication not indicated in files (*n* = 9, 3%). The effectiveness of PGE1 (defined by the reopening of the arterial duct and/or decrease of the pressure gradient <20 mmHg) was 92.6% (276/298 patients). Twenty-two patients had no haemodynamical or anatomical changes after PGE1 infusion.

At 5 years, 86.8% patients with PGE1 and 90.8% not receiving PGE1 at the time of aortic arch repair are free from reintervention (*P* = 0.008, Fig. 2).

In patients not receiving PGE1 (*n* = 232, 45.8%), freedom from reintervention was 93.3% beyond the first year, and there was no reintervention rate difference between extended end-to-side anastomosis and the classical end-to-end anastomosis (93.8% vs 90.9%, respectively, *P* = 0.69, Fig. 3). Only 2 patients (0.9%) needed a delayed reintervention beyond the first year.

When initial repair was performed with PGE1 (*n* = 298, 56.2%), freedom from reintervention was 88.7% after 1 year (*P* = 0.008, compared to patients not receiving PGE1). In addition, the freedom from reintervention rate was significantly different between extended end-to-side anastomosis and the classical end-to-end anastomosis (90% vs 83.4%, respectively, *P* = 0.033, Fig. 3). Seven patients (2.4%) needed a reintervention beyond 1 year of age (*P* = 0.023, compared to patients not receiving PGE1).

Age at repair

Two hundred and ninety-four (55.5%) patients had repair before 15 days of life and 103 (19.4%) patients between 30 and 90 days of life. At 5 years, 87.4% and 93.2%, respectively, are free from reintervention (*P* = 0.017, Fig. 4). Beyond the first year of life, no

patient who underwent surgery between 30 and 90 days of life needed reintervention on the aortic arch.

Risk factors of death

Overall mortality was 3.6% (19 patients). Ten patients died before hospital discharge: 5 patients in CoA, 4 patients in CoA + VSD and 1 patient in CoA + PAB. After discharge, mortality at reintervention was 1 for reintervention on the aortic arch and 3 for other reoperations. Five patients died after discharge from others causes: 2 septic shocks, 1 intra-alveolar haemorrhage, 1 pulmonary hypertension and 1 unknown cause.

Median age of death was 71 (9–879) days. Median delay between repair and death was 57 (0–858) days.

Overall mortality was 2.6% ($n = 10$) in isolated CoA, 7.8% ($n = 4$) in CoA + VSD and 5.3% ($n = 5$) in CoA + PAB ($P = 0.078$) (Fig. 5).

In multivariable analysis, the risk factors of death were the presence of VSD ($P = 0.037$) and low weight at initial repair ($P = 0.001$).

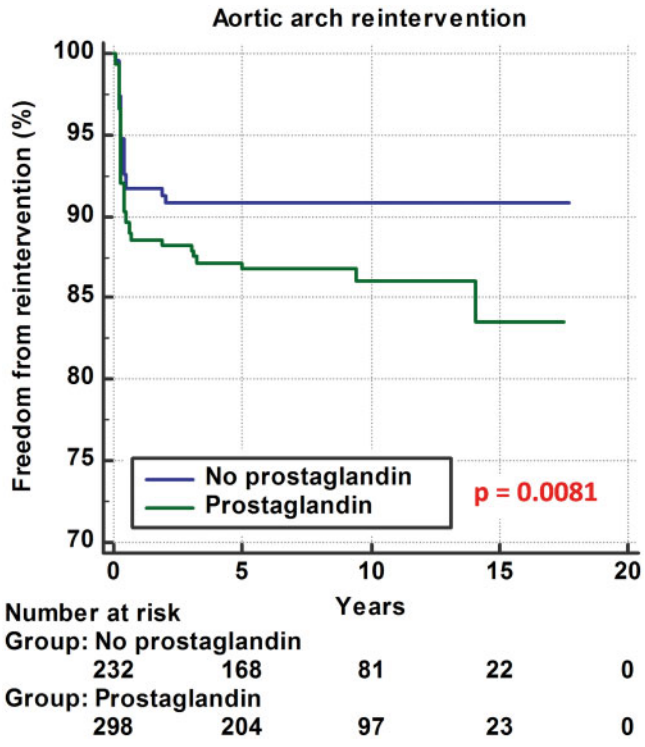

Figure 2: PGE1 infusion and risk of aortic arch reintervention (surgical and endovascular interventions).

DISCUSSION

Here, we show that clinical characteristics, anatomical parameters (including the anatomy of the aortic arch), as well as the type of surgical repair, were not associated with the risk of recoarctation in infants who had CoA repair before 3 months' age. Interestingly, PGE1 infusion and age at repair <15 days were the 2 factors associated with the risk of reintervention for recoarctation. These 2 factors are intrinsically linked as only neonates received PGE1 before CoA repair. Moreover, the technique of repair of the aortic arch (end-to-end or extended end-to-side) influenced the risk of recoarctation in children receiving PGE1. Indeed, reopening of the arterial duct changes the morphology of the aortic isthmus and may mask the boundaries of the zone to be resected. The decision to perform a classical end-to-end or an extended end-to-side anastomosis is usually taken during the operation. Our results suggest that a detailed analysis of the aortic arch anatomy should be more precise, particularly in children receiving PGE1 to improve the choice of surgical technique. Further, recoarctation occurred very early after repair in the majority of cases,

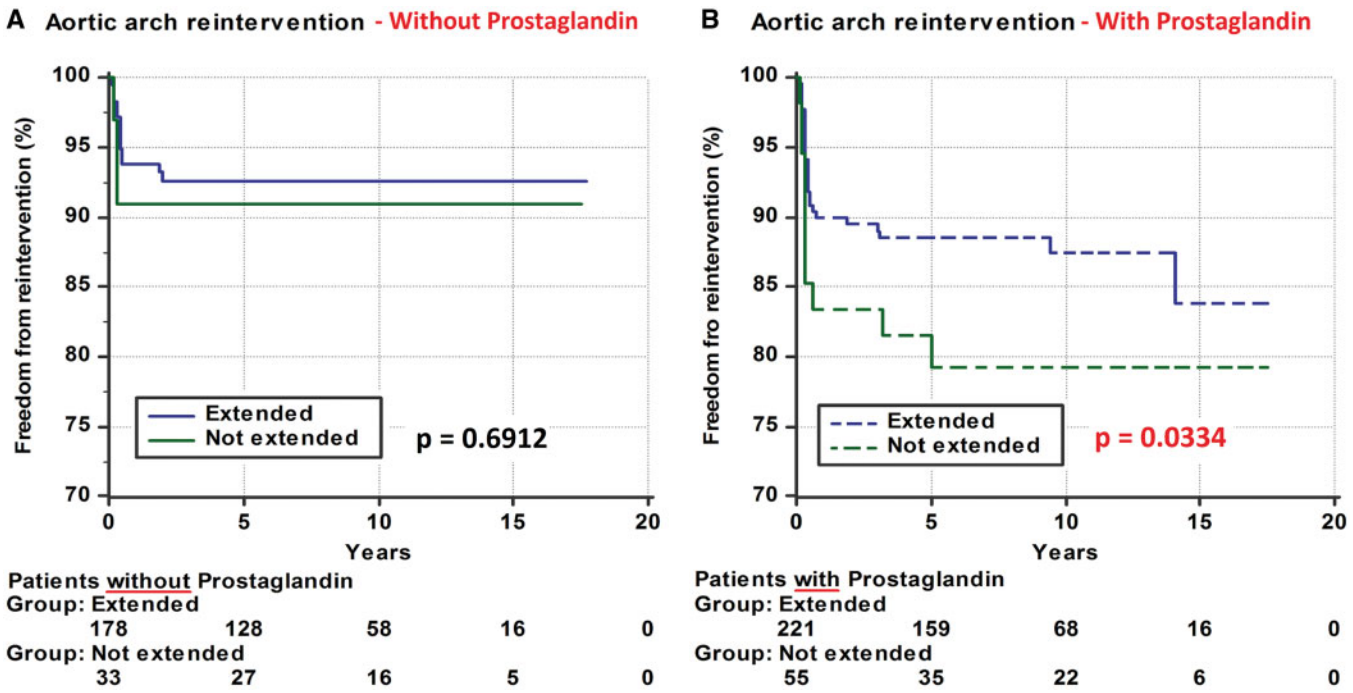

Figure 3: Comparison between extended and end-to-end repair in patients receiving PGE1 or not at time of surgical repair.

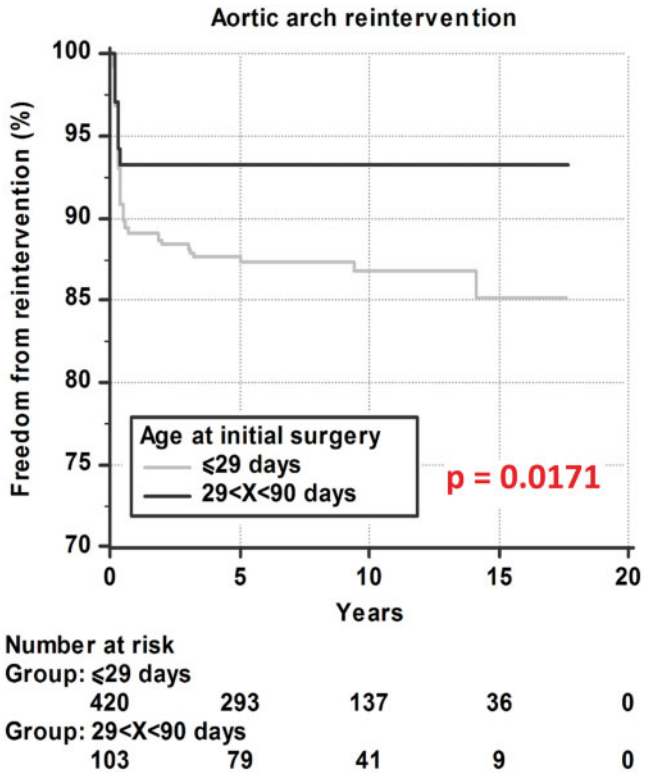

Figure 4: Age at repair and reintervention.

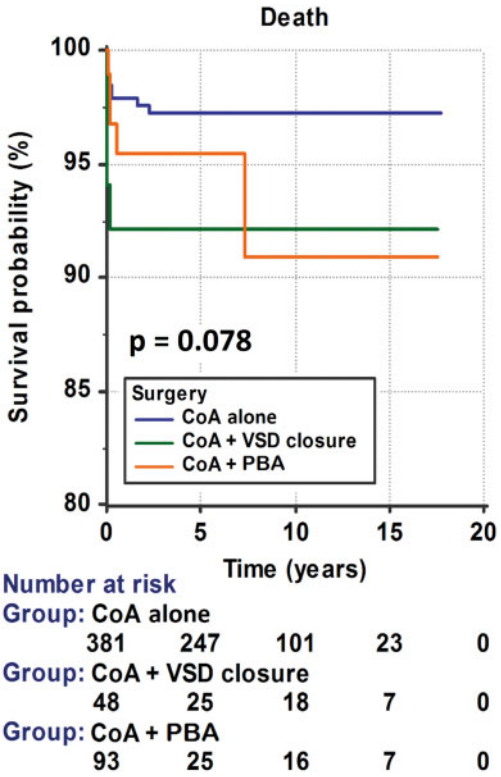

Figure 5: Mortality and surgical strategy.

suggesting the presence of residual ductal tissue within the aortic arch and incomplete remodelling before repair.

PGE1 is often necessary in children with complicated CoA to restore left ventricular function before surgery and to improve postoperative outcomes [12]. PGE1 reduces mortality while awaiting surgery, decreasing systemic resistance and allowing relaxation of the arterial duct or the aortic arch, although it has many haemodynamic and respiratory side effects [13, 14].

Liberman *et al.* [15] showed that treatment with prostaglandin also allowed reopening not only of the arterial duct but also of the CoA zone. It is of note that ductal tissue also extends in the aortic isthmus [16]. Actually, Russell *et al.* [17] showed an association between the recurrence of CoA and the presence of residual ductal tissue in the aorta. In 23 patients younger than 3 months of age who had CoA repair, histological examination of 22 specimens demonstrated a circumferential sling of ductal tissue that extended out from the arterial duct and surrounded the aorta at the level of the CoA shelf. Burch *et al.* [18] already found an association between PGE1 and reintervention. In this retrospective study of 167 patients, 14 (13.3%) of 105 patients receiving PGE1 for adequate perfusion underwent reintervention, whereas only 1 (2.4%) of 41 patients who were not PGE1 dependent underwent reintervention ($P = 0.07$). In a systematic review, Jonas [19] asked the question: do we need to resect ductal tissue facing a CoA? The underlying idea was to compare the 2 techniques used at the time: end-to-end anastomosis and subclavian flap aortoplasty. He finally concluded in favour of end-to-end anastomosis. Subsequently, in case of hypoplastic aortic arch, the various extended end-to-side strategies have shown that the wider the resection, the better the outcome, even including the left subclavian resection [10, 20–22].

The first possible strategy to decrease the risk of recoarctation could be to reduce the indications of PGE1 in neonates with CoA. Accordingly, Conte *et al.* showed that the prevalence of PGE1 therapy has increased from 40% in the period 1983–1989 to 96% in the period 1990–1994 ($P < 0.001$), without modification of the early outcomes. This study does not give clear information on the reasons to administrate PGE1 in this very high proportion of neonates. In our study, 62.4% of the patients receiving PGE1 had heart failure or a physiology mimicking interrupted aortic arch but the other patients had questionable indications.

Delaying the CoA repair is not possible in patients with reasonable indications to PGE1. Furthermore, it is largely accepted that surgery after 1 year exposes the patient to higher cardiovascular risks, including late systemic hypertension [23, 24]. However, there is limited available information on outcomes of patients operated on during their first year beyond the neonatal period. Mery *et al.* [25] compared children younger than 1 month ($n = 143$) with infants aged of 1–12 months ($n = 122$) who had aortic arch repair. They found a trend for the risk of reintervention in the youngest ($P = 0.06$). Wood *et al.* [9] in a series of 183 infants showed that all recoarctations were observed in patients operated before the age of 1 month. In our study, 103 patients underwent surgery after the age of 1 month and there was a significant difference in the reintervention rate compared to children operated on between 1 and 3 months ($P = 0.017$). These data suggest that delaying the surgery after 15 days or 1 month, when close follow-up is possible and when general status of the patient is excellent, may limit the risk of recoarctation potentially because the remodelling of the aortic arch is completed and the optimal resection of the abnormal zone of the aortic arch easier to define during surgery.

To obtain a better description of the aortic arch might be challenging when the arterial duct is open either with echocardiography or with computed tomography. In children under close

monitoring, transient stopping of the PGE1 infusion could help to better describe the anatomy of the aortic segments and guide surgical repair.

Limitations

While the number of infants was high in our study, the main limitation of our study is its retrospective and monocentric nature. In addition, it was not possible to precisely analyse the anatomy of each aortic segment in all cases to link the surgical technique that had been used with the exact morphological characteristics of the aortic arch. This certainly limited the identification of anatomical risk factors on top of those that we found.

CONCLUSION

The preoperative medical management of CoA with the use of PGE1 in neonates has a significant impact on the risk of recoarctation. The modalities of PGE1 use should be reconsidered to obtain an adequate description of the aortic arch segments to optimally guide surgical repair.

SUPPLEMENTARY MATERIAL

Supplementary material is available at *ICVTS* online.

Conflict of interest: none declared.

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