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Increased aortic wall stiffness is predictive of aortic dilation in adult patients following coarctation of the aorta repair

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ABSTRACT

Patients after repaired coarctation of the aorta are at risk for dilation and dissection of both the ascending and descending aorta. Histologic abnormalities seen are indistinguishable from patterns seen with Marfan syndrome, and lead to a stiff aortic wall, characterized by decreased distensibility, strain, and increased B-stiffness. We sought to analyze the relationship of aortic wall stiffness measured by cardiac MRI to aortic dilation following coarctation of the aorta repair. Fifty-two adult patients (26 females) with history of previous coarctation of the aorta repair who had undergone a recent cardiac MRI were identified. Studies and charts were reviewed retrospectively. Aortic strain, distensibility, and B-stiffness values were measured at level of ascending aorta and descending aorta. Relationships between measurements of aortic stiffness were compared to aortic dimensions. Median age at coarctation repair was 0.2 years (range 0.1–36 years). Types of repair included subclavian artery flap (44%), end-to-end anastomosis (33%), and patch angioplasty (15%). Time from repair to cardiac MRI was 25 years (range 1–42). By cardiac MRI, 38% had a dilated ascending aorta defined as a Z-score of > 2.5. On average the ascending aorta was stiffer than the descending aorta for all three parameters. For both the ascending and descending aorta the 3 parameters of aortic stiffness were predictive of increasing aortic dimensions (P value < 0.01). By univariate analysis, risk factors for a dilated ascending aorta defined as a Z-score of > 2.5 included older age at the time of initial repair, presence of a bicuspid aortic valve, and worse parameters of ascending aorta stiffness: % strain, distensibility, and B-stiffness, and a lower descending aorta % strain. By multivariate analysis, older age at initial repair and both a lower % strain at both the ascending and descending aorta remained statistically significant.

1. Introduction

The propensity for dilation and dissection of both the ascending and descending aorta in patients following coarctation of the aorta repair has long been recognized [1]. Type of surgical repair, age at initial repair, presence of residual obstruction, and systemic hypertension have been associated with late aneurysm formation [1,2]. Analysis of the aortic walls of these patients demonstrate elastic fiber fragmentation, smooth muscle cell apoptosis, with collagen deposition [1,3]. These findings are indistinguishable from the pattern seen in the walls of aneurysm in patients with Marfan syndrome, bicuspid aortic valves, and other forms of familial aortopathies [3]. In addition, while these findings progress over time, they can be identified early in life, even as a neonate [4]. Early studies have demonstrated that these changes lead to a stiff aortic wall characterized by decreased distensibility, as measured by cardiac magnetic resonance imaging (CMR) [5]. Numerous studies have demonstrated that wall distensibility is crucial to the function of the aorta; not only does the aorta serve as a conduit to carry blood to the peripheral branches, but the aorta serves as a vascular buffer to each ventricular contraction [6,7]. This ability is dependent on the distensibility of the aortic wall, which correlates with the elastic fiber content of the wall, and is clearly altered in the setting of aneurysm formation [6]. Over the last several years, a growing body of evidence has been published supporting the hypothesis, that aortic

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stiffness is a major player in aortic aneurysm formation and that aortic stiffness does not result from dilation but rather exists before dilation and is a major determinant of progressive dilation and risk for dissection [1,6]. There is limited data looking at aortic wall stiffness following coarctation of the aorta repair. Small studies have demonstrated abnormal wall stiffness and vascular reactivity, but no studies have been performed looking at the relationship of aortic wall stiffness and aortic dilation. The objective of our study was to analyze the relationship of aortic wall stiffness with aortic dilation in patients following coarctation of the aorta repair.

2. Methods

2.1. Subjects

After obtaining IRB approval, 52 adult patients with history of previous repair of simple coarctation of aorta who had undergone a recent cardiac MRI and were being followed by our adult congenital program were identified. Simple coarctation was defined as a discrete stenosis of the proximal thoracic aorta without other hemodynamic significant lesions: ventricular septal defects, left ventricular outflow tract obstruction, mitral valve stenosis. A retrospective review of existing clinical data was then performed.

2.2. Cardiac MRI

Clinically indicated CMR scans were performed on 1.5 T or 3 T Siemens magnets (Avanto®, Symphony®, and Skyra®, Siemens Medical Solutions USA, Malvern PA), according to standard clinical imaging protocol to determine ventricular volumes and systolic function, assess post-operative aortic arch anatomy, and to complete quantification of vascular blood flow. Electrocardiogram-gated 2-dimensional cine steady state free precession (SSFP) or fast low angle shot (FLASH) imaging of the aortic arch and left ventricular outflow tract (LVOT) was performed. Using two orthogonal long axis planes of the LVOT that were then used to prescribe a stack of slices in the short-axis of aortic root and ascending aorta. After placement of a peripheral intravenous cannula, 0.4 mL/kg of gadolinium (gadodiamide, Omniscan) at a rate of 2 mL/s. Magnetic resonance angiography (MRA) image acquisition was initiated using bolus tracker to optimize visualization of the aorta. Utilizing the prior imaging for planning, free breathing phase contrast velocity encoded through-plane CINE imaging was obtained (FOV 256 × 192, slice thickness 5–6 mm, bandwidth 390, TE 3.26–4.1, TR 27.75–46, ETL 1, NEX 3) in the ascending aorta and descending aorta (Fig. 1).

2.3. Cardiac MRI image analysis

Phase contrast CINE CMR images were then post processed off line using Circle CVI 42 software at the level of the ascending aorta (AAo) and thoracic descending aorta (DAo) (Fig. 1). At each location, the aorta was traced to determine the systole and diastole. Based from the aortic flow curve, systolic and diastole were identified and the aortic diameter was measured. From the systolic and diastolic diameters, cross-sectional area was determined and the following parameters of aortic stiffness were calculated as previously described [13].

$$\text{Strain} = \frac{\text{Systolic area} - \text{Diastolic Area}}{\text{Diastolic area}}$$

$$\text{Distensibility} = \frac{\text{Strain}}{\text{Brachial pulse pressure}}$$

$$\text{Beta stiffness index} = \frac{\log(\text{systolic BP}/\text{diastolic BP})}{\text{Strain}}$$

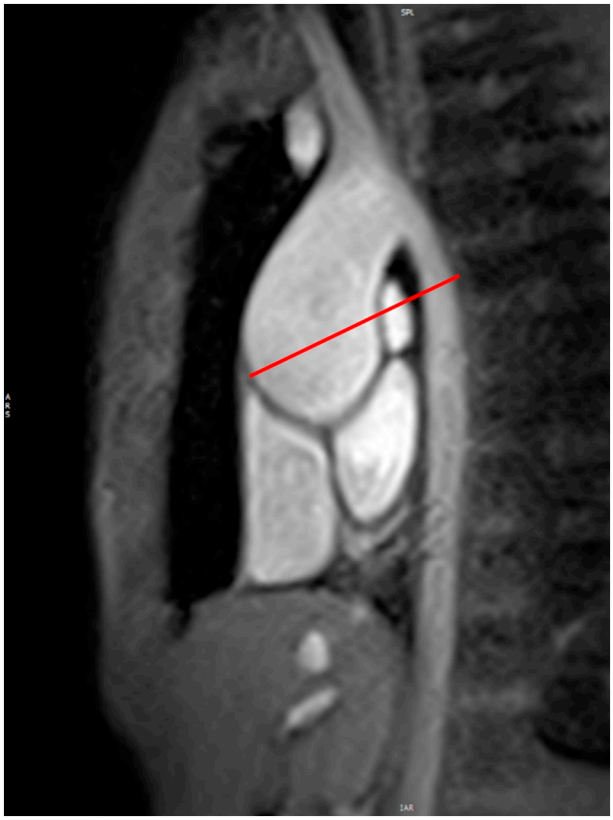

Fig. 1. Measurement of aortic root stiffness by CMR. Phase contrast CINE CMR images were processed off line using Circle CVI 42 software at the level of the ascending aorta and thoracic descending aorta. At each location, the aorta was traced to determine the systole and diastole. Based from the aortic flow curve, systolic and diastole were identified and the aortic diameter was measured. From the systolic and diastolic diameters, cross-sectional area was determined and the parameters of aortic stiffness were calculated. Red line = level of measurements.

2.4. Statistical analysis

Spearman correlation coefficients were used to assess for associations between stiffness parameters and aortic size at each of the anatomic levels. Univariate associations between dilation at the ascending aorta and clinical variables were examined using the Student's *t*-test, Wilcoxon-Mann-Whitney test or Fischer's exact test, as appropriate. Multivariate logistic regression modeling with forward selection was performed to identify predictors of ascending aorta dilation. Variables demonstrating univariate associations with P value < 0.05 qualified for inclusion into the final model. For all analyses, statistical significance was evaluated with respect to a type 1 error probability threshold of 0.05.

3. Results

3.1. Subjects

The characteristics of our cohort are shown in Table 1. For our cohort, half the patients were women. The mean age at repair was 0.2 years, with a range of 0.1 to 36 years. The majority of patients had undergone previous repair using the subclavian artery flap technique, accounting for 44% of the cohort. An additional 33% of patients had undergone repair using resection with end to end anastomosis, 15% previous patch angioplasty and 8% had an extended end to end anastomosis. A total of 77% of our cohort had bicuspid aortic valves (40/52).

Table 1
Patient characteristics of cohort.

Female	26	(50%)
Mean age at repair (years)	0.2	(0.1–36)
Type of repair		
Subclavian artery flap	23	(44%)
End to end anastomosis	17	(33%)
Patch angioplasty	8	(15%)
Extended end to end anastomosis	4	(8%)
Bicuspid aortic valve	40	(77%)
Aortic valve intervention	0	
Reintervention for residual/recurrent arch obstruction	9	(17%)
Balloon angioplasty	7	
Stent placement	2	
Hypertension at last follow-up	17	(33%)
ACE or ARB	11	
Beta blocker	8	
Both	5	
Smoking	4	(8%)
Hyperlipidemia	3	(6%)
Statin therapy	3	
Mean systolic BP	122	(Range 90–178)
Mean diastolic BP	40	(Range 20–64)
Mean arm to leg systolic BP gradient (mm Hg)	6 ± 7	(Range 0–19)
Mean time from repair to cardiac MRI (years)	25	(Range 1–42)
Mean ascending aorta size (cm)	3.2	(Range 2.2–5.4)
Mean ascending aorta Z-score	2.4	(Range 0.8–8.1)
Ascending aorta Z-score > 2.5	20	(33%)
Ascending aorta Z-score > 4.0	9	(17%)
Mean descending aorta size (cm)	2.0	(Range 1.5–3.8)

Table key: ACE = ace inhibitor, ARB = angiotensin II blocker, BP = blood pressure. cm = centimeters, mmHg = millimeters of mercury.

Mean time from initial repair to cardiac MRI was 25 years, with a range of 1 to 42 years. Mean age at time of last follow-up was 27 years (range 16–51). At time of cardiac MRI, 17% (9/52) of the cohort have required reintervention for residual arch obstruction, with majority having undergone balloon angioplasty (78%, 7/9) and 2 having undergone balloon dilation with stent placemen. No patient in the cohort has required aortic valve intervention or ascending aorta replacement. At time of cardiac MRI, 33% of the patients (17/52) were being treated for hypertension. Eleven patients were taking an Ace inhibitor or an angiotensin II blocker, 8 a beta blocker, and 5 both. Four patients in the cohort were actively smoking and 3 were being treated for hyperlipidemia with a statin.

Overall, 20 (38%) patients were found to have a dilated ascending aorta defined as an ascending aortic Z score of > 2.5. Of these 20 patients, 9 (17%) had moderate to severe ascending dilation with a Z-score of > 4.0. The mean ascending aorta diameter for the cohort was 3.2 cm (range 2.2–5.4 cm). The mean ascending aorta Z-score was 2.4 (range 0.8–8.1) The max descending aorta diameter was 2.0 cm (range 1.5–3.8 cm).

3.2. Aortic stiffness and associations

The parameters of aortic stiffness at the 2 locations are summarized in Table 2. For % wall strain and wall distensibility, lower values are indicative of a stiffer aorta, while for B-stiffness index, a higher value denotes a stiffer aorta. On average the ascending aorta was stiffer than the descending aorta for all three parameters.

Table 2
MRI measurements of aortic wall stiffness.

	AAo	DAo
Wall strain %	20 (0.11–56)	29 (5.5–64)
Wall distensibility (X 10^{-3} mm Hg^{-1})	4.2 (0.7–14)	4.3 (1–9.3)
β-Stiffness index	4.1 (0.8–18.7)	3.0 (0.4–8.6)

Table key: % = percentage, AAo = ascending aorta, DAo = descending aorta, mm Hg = millimeters of mercury.

For the ascending aorta (Fig. 2) and for the descending aorta (Fig. 3), aortic size increased as all 3 parameters of stiffness worsened. Interestingly, when we compared the parameters of descending aorta stiffness we found similar results, with ascending aorta size increasing as all 3 parameters of descending aorta stiffness worsened (Fig. 4).

To further delineate the relationship of the parameters of aortic stiffness to ascending aorta size, we performed univariate and multivariable linear regression analysis (Table 3). By univariate analysis, risk factors for a dilated ascending aorta defined as a Z-score of > 2.5 included older age at the time of initial repair, presence of a bicuspid aortic valve, and worse parameters of ascending aorta stiffness: % strain, distensibility, and B-stiffness, and a lower descending aorta % strain. By multivariate analysis, older age at initial repair and both a lower % strain at both the ascending and descending aorta remained statistically significant.

4. Discussion

In this study we found that CMR derived parameters of aortic stiffness at both the ascending aorta and the descending aorta correlated with aorta size at each level. Interestingly, we also found that parameters of aortic stiffness at the descending aorta also correlated with ascending aorta size. This supports findings of others that coarctation of the aorta is not a simple mechanical obstruction but is associated with diffuse aortic arch wall remodeling and long-term abnormalities of vascular dysfunction regardless of successful surgical repair [9–11]. In a series of studies by Ou et al., patients following coarctation of the aorta repair had decreased ascending aorta distensiblity coupled with increased loss of systolic wave amplitude across the aortic arch with an associated risk for resting and exercise induced hypertension together with left ventricular hypertrophy [15–18]. In a study by de Divitiis and colleagues, adult patients following coarctation of the aorta, (mean age 19 ± 10 years) had abnormal vascular reactivity as measured by flow mediated dilation and nitroglycerin challenge along with echo derived measurements of aortic stiffness even late after successful repair (absence of residual obstruction) [5]. More recently, Wegner and colleagues demonstrated using CMR for pulse wave computation in young adolescents with history of surgical repair of coarctation of the aorta that elevated stiffness was not isolated to either the ascending or descending aorta but was present throughout the thoracic aorta [8].

The fact that both ascending and descending parameters of stiffness correlated with aortic size in our study is further supported by previous pathology series, demonstrating similar histologic changes in both the ascending and descending aorta in neonates with coarctation of the aorta [4]. This was confirmed early on in a study by Schested et al. In this study, the authors demonstrated an increase in collagen content and a decrease in smooth muscle content in the aortic segment above the coarctation segment in samples of neonates undergoing coarctation of the aorta repair [10]. These findings are indistinguishable from the findings seen in the ascending aorta of patients with bicuspid aortic valves and other forms of familial aortopathies [1,3].

The fact that these histologic changes occur early in life, likely explain why we found in this study that in addition to aortic stiffness parameters, later age at initial repair of coarctation of the aorta was also predictive of ascending aorta dilation. In a study by Oliver and colleagues, older age at repair was associated with aneurysm formation with a cutoff in this study of 13.5 years [19]. Previous studies have also suggested that earlier repair of coarctation of the aorta may be associated with better preserved elastic properties of the aorta. In the previous mentioned study by de Divitiis et al., patients who underwent repair of their coarctation of the aorta in the first 4 months of life had improved arterial stiffness compared to those repaired later [5]. This is further supported by the study published by Vogt and colleagues. In their study, they found that the elastic properties of the prestenotic

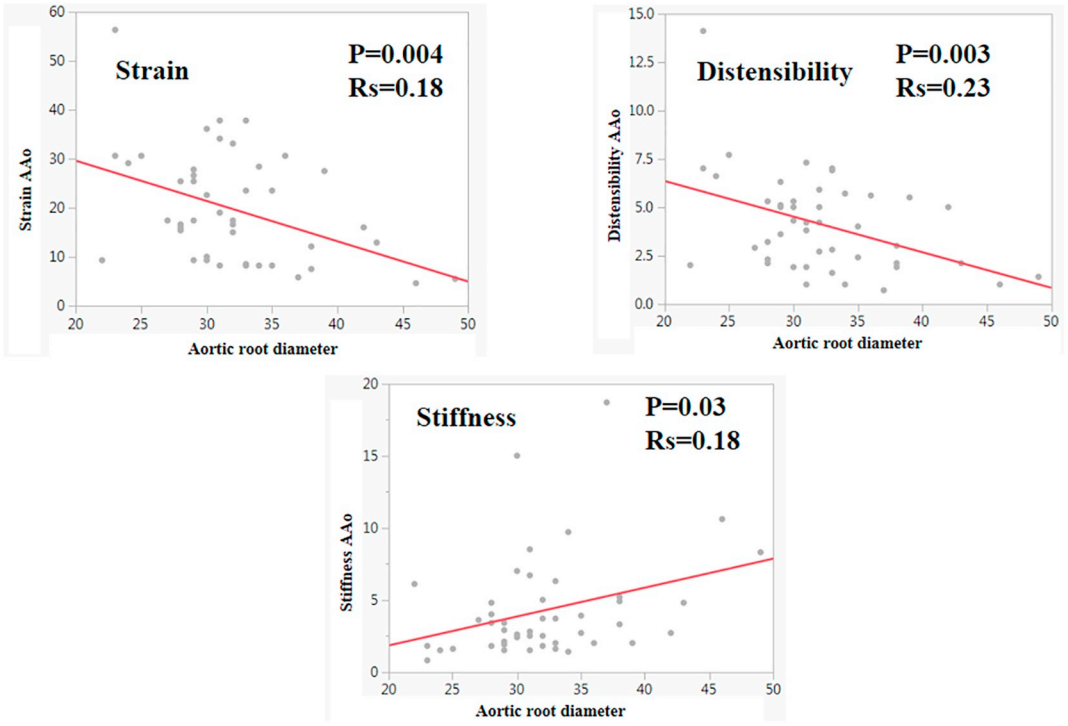

Fig. 2. Correlation between ascending aorta diameter and parameters of ascending aortic wall stiffness. As parameters of the ascending aortic wall stiffness worsen the ascending aorta size increases. For % wall strain and wall distensibility, lower values are indicative of a stiffer aorta, while for B-stiffness index, a higher value denotes a stiffer aorta. AAo = ascending aorta.

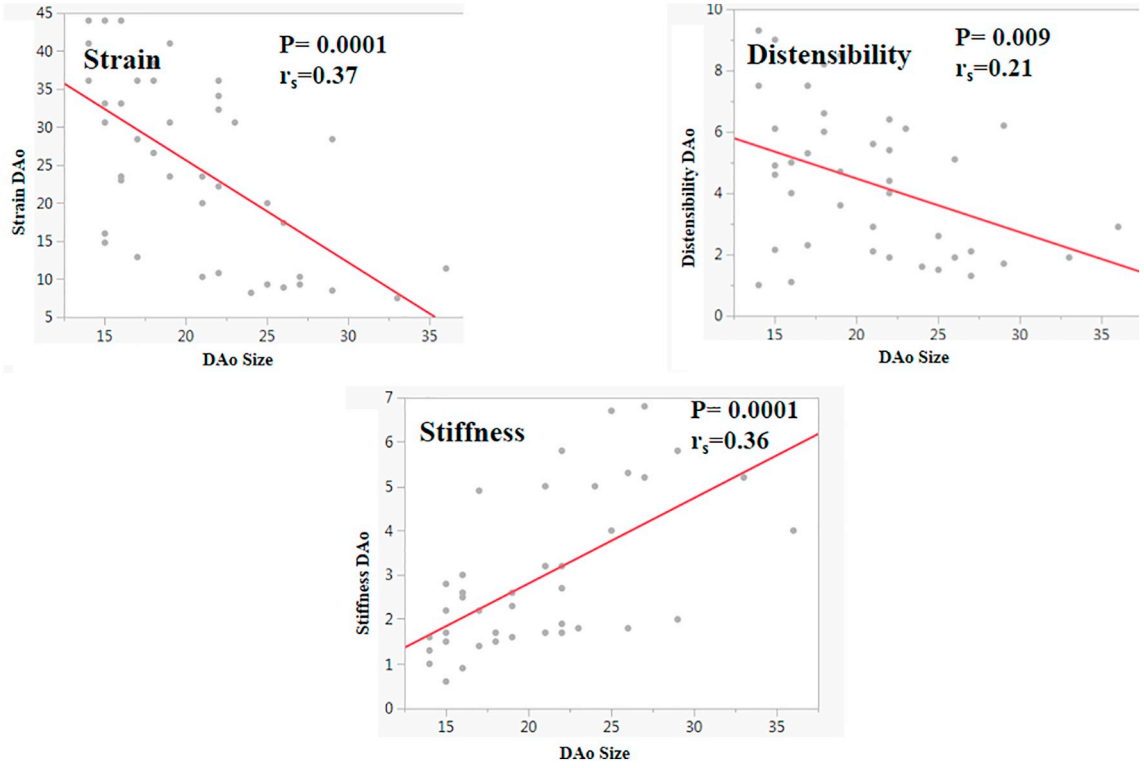

Fig. 3. Correlation between descending aorta diameter and parameters of descending aortic wall stiffness. As parameters of descending aortic wall stiffness worsen, descending aorta size increases. For % wall strain and wall distensibility, lower values are indicative of a stiffer aorta, while for B-stiffness index, a higher value denotes a stiffer aorta. DAo = descending aorta.

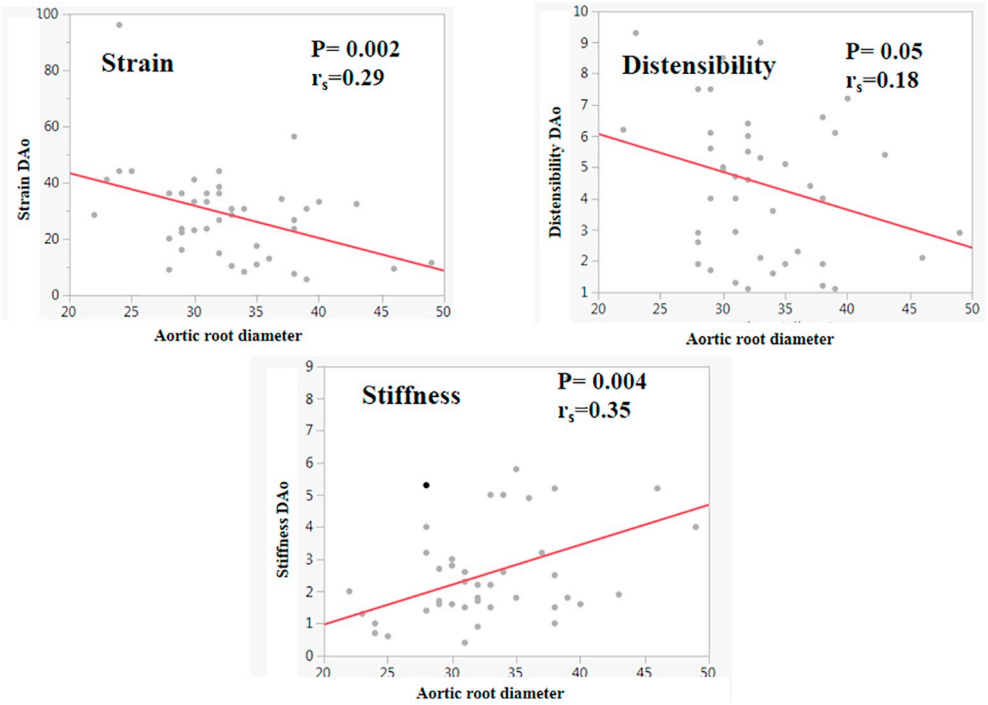

Fig. 4. Correlation between ascending aorta diameter and parameters of descending aortic wall stiffness. As parameters of descending aorta wall stiffness worsen, the ascending aorta size increases. For % wall strain and wall distensibility, lower values are indicative of a stiffer aorta, while for B-stiffness index, a higher value denotes a stiffer aorta. DAo = descending aorta.

Table 3
Risk factors associated with ascending aorta dilation (Z-score > 2.5).

Parameter	AAo Z-score > 2.5 *N* = 20	AAo Z-score < 2.5 *N* = 32	Univariate *P*-value	Multi *P*-value	Odds ratio	95% CI
Older age at repair (yrs)	6 ± 1.5	1 ± 1.1	0.05	0.01	1.8	1.4–2.4
Bicuspid aortic valve	94%	61%	0.006	0.06	0.14	0.1–0.6
Ascending aorta % strain	21.8 ± 1.8	11.5 ± 3.0	0.02	0.03	1.2	0.9–1.4
Ascending aorta distensibility	2.6 ± 0.8	4.6 ± 0.4	0.04	0.1	0.6	0.3–1.2
Ascending aorta stiffness	7.0 ± 1.2	3.6 ± 0.5	0.02	0.08	0.7	0.4–1.14
Descending aorta % strain	19.2 ± 3	27.8 ± 2.2	0.03	0.01	1.1	0.9–1.3
Descending aorta distensibility	4.3 ± 0.5	4.4 ± 0.6	NS			
Descending aorta stiffness index	3.3 ± 0.4	2.6 ± 0.3	NS			
Length of follow-up (yrs)	27 ± 2.1	25 ± 1.7	NS			
Re-intervention	20%	16%	NS			
Hypertension	30%	36%	NS			
Medication use	53%	46%	NS			
Ace/Arb	22%	14%	NS			
Beta blocker	18%	17%	NS			
Type of repair	–	–	NS			

Table key: ACE = ace inhibitor, AA0 = ascending aorta, ARB = angiotensin II blocker, CI = confidence interval, multi = multivariate, NS = not significant, % = percentage, yrs = years.

aorta of neonates with coarctation were already abnormal and importantly, did not improve in the short term after surgical repair [4].

In our study, not surprising, the presence of a bicuspid aortic valve was a risk factor for ascending aorta dilation following coarctation of the aorta repair. In a series by Oliver and colleagues, the presence of a bicuspid aortic valve was an independent risk factor for both ascending and descending aortic aneurysm formation [19]. In addition, previous studies have demonstrated abnormal aortic stiffness and distensibility to be common in patients with bicuspid aortic valves and have been associated with worsening aortic regurgitation and LVH [20,21]. Similarly, in a recent study by Schäfer and colleagues, in children following coarctation of the aorta repair, the presence of a bicuspid aortic valve was associated with worst parameters of aortic stiffness compared to those with a tricuspid aortic valve [9].

It is important to mention that we did analyze impact of several other variables that have been reported previously to affect aortic size and stiffness following coarctation of the aorta repair and found no correlation in our cohort [4,19]. Coarctation of the aorta repair using patch angioplasty is a well-known risk factor for aneurysmal formation in the proximal descending thoracic aorta [2]. At same time, previous studies have demonstrated type of coarctation of the aorta repair as a risk factor for increased aortic stiffness [19]. In a study by Schäfer and colleagues, children with coarctation of the aorta whom had underwent surgical repair or balloon angioplasty, had increased ascending aortic stiffness when compared to normal controls. Interestingly, in this same study, aortic stiffness in children, whom had been treated with stent alone however, was not statistically different compared to controls [9]. In our study, we did not find a significant difference between the different surgical techniques and aortic size and stiffness. The cohort in our study however, was much older (mean age 27 years verse 13 years) with much longer follow-up (25 years verse 13 years) which could explain this observational difference between the two studies. In our

study we also did not find hypertension as a risk factor for aortic dilation. Systemic hypertension is a major long-term complication after coarctation of the aorta repair [22,23]. Data in hypertensive adults, have demonstrated a strong correlation between systemic hypertension and aortic wall stiffness, and the presence of LVH, aortic dilation, and risk of morbidity and mortality [7,24]. The question left unanswered is whether aortic stiffness is a consequence of systemic hypertension or if aortic stiffness leads to risk of hypertension [15,16]. Further studies are required to analyze the associated between aortic stiffness, hypertension, and aortic size.

5. Study limitations

Our study does have several limitations. This was a retrospective study at a single center as result, we are subject to referral bias. In addition, the BP measurements in our study were not always measured immediately pre and post CMR acquisition introducing potential for altered aortic stiffness metrics. Our study is also limited by factors inherent to the calculations used in measuring aortic stiffness with CMR. Measurements of peripheral systolic blood pressures have been shown in previous studies to be non-reflective of central aortic pressure [6]. BP waveforms obtained by catheterization would enable more accurate analysis of central and systemic arterial stiffness using impedance spectra evaluation, yet this approach would add an additional component of invasiveness which does limit the use of this technique. MRI based measurements of aortic stiffness at this point remain manual and lack full automatization and as result, are subject to intra-observer variability.

6. Conclusion

The results of our study show that CMR determined parameters of aortic wall stiffness correlate with both ascending and descending aorta dimensions in adult patients following coarctation of aorta repair. With increased aortic dimensions, parameters of aortic stiffness worsen. Other predictors of ascending aorta dilation include the presence of a bicuspid aortic valve and older age at time of initial coarctation of repair. Our data supports the need for further studies to access whether parameters of the ascending aortic and descending aortic stiffness are predictive of progressive dilation and dissection over time and to further evaluate the effect of medical therapy in efforts to improve aortic stiffness and prevent the late complications of aortic dilation and risk for dissection.

Conflict of interest

We wish to confirm that there are no known conflicts of interest associated with this publication and there has been no significant financial support for this work that could have influenced its outcome.

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