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ORIGINAL ARTICLE

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Coarctation repair normalizes left ventricular function and aorto-septal angle in neonates

Haki Jashari, MD[1] | Katarina Lannering, MD[2] | Mats Mellander, MD, PhD[2] | Pranvera Ibrahimi, MD, PhD[1] | Annika Rydberg, MD, PhD[3] | Michael Y. Henein, MD, PhD[1]

[1]Department of Public Health and Clinical Medicine, Umeå University, Umeå, Sweden

[2]Department of Pediatric Cardiology, Queen Silvia Children's Hospital, Sahlgrenska University Hospital, Göteborg, Sweden

[3]Department of Clinical Sciences, Umeå University, Umeå, Sweden

Correspondence
M.Y. Henein, Department of Public Health and Clinical Medicine, Umeå University, and Heart Centre, Umeå, Sweden.
Email: michael.henein@umu.se

Abstract

Background and aims: Patients with coarctation of the aorta (CoA) have increased left ventricular (LV) afterload that has been shown to impact the LV and ascending aortic function. We aimed to examine the effect of coarctation on LV function and aorto-septal angle (AoSA) before and after surgical repair.

Methods: We retrospectively studied 21 patients with surgically repaired CoA at a median age of 9 (2-53) days at three time points: (1) just before intervention, (2) at short-term follow-up, and (3) at medium-term follow-up after intervention. AoSA was measured from the parasternal long axis view, at three time points during the cardiac cycle: (1) end diastole, (2) beginning of systole, and (3) at peak ejection in the descending aorta. In addition to conventional LV structure and function, global longitudinal strain, and strain rate were measured using STE technique and Tomtec software. Three groups of age matched healthy children served as controls at each time point.

Results: AoSA was significantly wider before intervention, in particular at peak ejection in the descending aorta ($144° \pm 6.4°$ vs. $136° \pm 4.1°$; $P < .0001$), and correlated with CoA pressure gradient. After intervention, AoSA normalized and significantly correlated with the increase of LV cavity function and overall LV deformation parameters.

Conclusions: AoSA is abnormally wide in neonates with CoA and is associated with severity of obstruction, LV dysfunction and compromised LV global deformation.

KEYWORDS
aorto-septal angle, coarctation of the aorta, left ventricular function, neonate

1 | INTRODUCTION

Coarctation of the aorta (CoA) is a congenital anomaly, whereby a narrowing of the aorta restricts blood flow and increases backward pressure on the left heart. This pressure afterload has been shown to impact the left ventricular (LV) cavity function as well as overall and regional myocardial function.[1,2] An important component of the LV is the outflow tract which demonstrated certain abnormalities in patients with other forms of pressure afterload, for example, aortic stenosis.[3–5] In particular, the aorto-septal angle (AoSA), which is part of the LV outflow tract (LVOT), has been shown to be abnormally wide in such patients.[5] Whether similar abnormalities exist in CoA, remain fixed or normalize after removal of pressure afterload remains to be determined.

The AoSA can easily and accurately be studied by two-dimensional echocardiography. We therefore sought to better understand the impact of increased afterload due to CoA on LV structure and function, particularly on the outflow tract and its relationship with the AoSA, in children and the effect of pressure afterload correction.

2 | METHODS

2.1 | Study population

We retrospectively studied 21 consecutive patients, with corrected CoA and median age at diagnosis of 4 (1–42) days, who were followed up at the Umeå University Hospital, Umeå and Queen Silvia Children's

TABLE 1 General characteristics of patients

Variable	
No *(%)*	21 (100)
Sex (Males) *n (%)*	14 (67)
Gestational weeks	39.9 ± 1
Birth weight, g	3506 ± 636
Birth length, cm	49.7 ± 2.9
BAV *n (%)*	10 (48)
Age at operation, days	9 (2-53)
Type of intervention *(n)*	ETE (19), SF (2)

BAV, bicuspid aortic valve; ETE, end-to-end anastomosis; SF, subclavian flap.

Hospital, Göteborg, Sweden, between 2006 and 2012 (Table 1). A complete history (age, gender, birth weight, age at intervention, type of intervention, follow-up duration, associated syndromes), physical examination (blood pressure, heart rate, weight and height/length) (Table 2), and echocardiographic recordings were analyzed at three time intervals: (1) just before intervention, (2) at intermediate follow-up (within the first 3 months after intervention), and (3) at medium-term follow-up after intervention (median 2.3 [0.57–4.13] years) (Table 3). Exclusion criteria were any associated congenital heart disease (except BAV), suboptimal echocardiographic windows or missing echocardiographic recordings at any of the three time points. Data were compared with age matched healthy children at each follow-up event.

2.2 | Echocardiographic data acquisition

Philips Healthcare and General Electric ultrasound machines were used to obtain all echocardiograms from Umeå and Göteborg, respectively. The same echo system was used at follow-up, except in three patients who were examined in two centers and had shared echo images. Peak systolic flow velocity at the proximal descending thoracic aorta was measured from the suprasternal window with continuous wave Doppler velocities, and the pressure drop (gradient) was calculated using the modified Bernoulli equation (pressure gradient = $4V^2$). LV cavity dimensions were measured using M-mode echocardiography in the parasternal long-axis view according to the recommendations of the European Society of Cardiology[6]; the ejection fraction and fractional shortening were then calculated. LVOT and aortic root (AoR) diameter at the level of the sinuses of Valsalva were measured according to the recommendations of the American Society of Echocardiography.[7] Aortic arch hypoplasia was identified if proximal aortic arch diameter, or distal aortic arch diameter, or aortic isthmus diameter were 60%, 50%, 40%, or less than the diameter of the ascending aorta, respectively.[8]

Aorto-septal angle (AoSA), the angle between the long axis of the ascending aorta and the plane of the interventricular septum (Figure 1),[9] was measured from the frozen 2D images of the parasternal long axis view. The midline axis of the aortic root was drawn from its mid-diameter at the level of the base of the aortic valve leaflets to that of the sinotubular junction. The midline axis of the septum was drawn from the midseptal point at the level of the mitral leaflet tips to the midseptal point 2 cm more apically (Figure 1). The angle between the two axes was measured offline by OsiriX (version 7.5), an image processing software dedicated to Digital Imaging and Communications in Medicine (DICOM) images. All AoSA measurements were made at three time points in the cardiac cycle; (1) just before mitral valve closure (end-diastole), (2) just after aortic valve opening (beginning of systole), and (3) at peak ejection in the descending aorta.

LV longitudinal myocardial deformation function was studied from the apical 4-chamber view using STE technique, and offline

TABLE 2 Demographics and clinical examination data

	Before intervention		Short-term postintervention		Medium-term postintervention	
	CoA (21)	Controls (20)	CoA (21)	Controls (20)	CoA (21)	Controls (20)
Age, d	4 (1-42)	6 (1-45)	20 (7-126)	32 (6-148)	880 (213-1510)	695 (421-1465)
Age postintervention, d	–	–	8 (2-97)	–	850 (208-1508)	–
Sex (males) *n (%)*	14 (67)	12 (60)	14 (67)	14 (70)	14 (67)	14 (70)
Gestational weeks	39.9 ± 1	39.2 ± 1	–	–	–	–
Weight, kg	3.5 ± 0.6	3.7 ± 0.4	–	3.9 ± 0.5	13.3 ± 2.9	14.1 ± 2.0
Length/height, cm	50 ± 2.9	52.1 ± 2.1	–	52.5 ± 3	90.4 ± 9.6	94.3 ± 10.7
BSA, m^2	0.2 ± 0.02	0.2 ± 0.01	–	0.2 ± 0.02	0.7 ± 0.2	0.7 ± 0.2
HR	142 ± 12	140 ± 15	130 ± 14	136 ± 10	106 ± 13	112 ± 9
SBP arm, mm Hg	88.4 ± 18	–	–	–	101 ± 10	–
DBP arm, mm Hg	54.4 ± 15.5	–	–	–	61 ± 8	–
SBP leg, mm Hg	64.8 ± 19.7	–	–	–	114 ± 15	–
DBP leg, mm Hg	42.8 ± 15.6	–	–	–	60 ± 13	–

d, days; BSA, body surface area; HR, heart rate; SBP, systolic blood pressure; DBP, diastolic blood pressure.
Comparison was not conducted where not applicable due to maturational changes.

TABLE 3 Conventional echocardiography data

	Before intervention (BI)		*P*	Short-term post-intervention (STPI)		*P*	Medium-term post-intervention (MTPI)	
	CoA (21)	Controls (20)	BI vs. STPI	CoA (21)	Controls (20)	STPI vs. MTPI	CoA (21)	Controls (20)
PG, mm Hg	40 (27-121)	–	<0.001	17 (5-31)	–	NS	18 (5-38)	–
EF %	55.1 ± 15.9[a]	68 ± 7.2	<0.01	65.7 ± 9.6[a]	68 ± 7	NS	69.7 ± 6.4	67.8 ± 5.2
FS %	27 ± 10.5[a]	35.2 ± 4.9	<0.01	34.4 ± 7.2[a]	34.7 ± 4.3	NS	37.1 ± 3.6	36.6 ± 3.1
LVESD, cm	1.3 ± 0.3	1.3 ± 0.2		1.2 ± 0.2	1.2 ± 0.1		1.9 ± 0.3	2.1 ± 0.4
LVEDD, cm	1.7 ± 0.3	1.8 ± 0.2		1.8 ± 0.3	2.0 ± 0.3		3.1 ± 0.3	3.3 ± 0.3
IVSs, cm	0.5 ± 0.1	0.5 ± 0.1		0.6 ± 0.1	0.6 ± 0.2		0.8 ± 0.2	0.7 ± 0.1
IVSd, cm	0.4 ± 0.1	0.4 ± 0.1		0.5 ± 0.1	0.6 ± 0.1		0.5 ± 0.1	0.5 ± 0.1
LVPWs, cm	0.5 ± 0.1	0.5 ± 0.2		0.6 ± 0.1	0.6 ± 0.2		0.8 ± 0.2	0.8 ± 0.1
LVPWd, cm	0.4 ± 0.1	0.4 ± 0.1		0.4 ± 0.1	0.5 ± 0.2		0.5 ± 0.1	0.5 ± 0.2
LV mass, g	10 ± 3.7	9.7 ± 3.5		12.6 ± 4.2	13.5 ± 3.6		34.8 ± 10.4	35.1 ± 12.1
LVMI, g/m$^{2.7}$	65.5 ± 26	64 ± 19.5			58.9 ± 22		48.5 ± 12.5	43.2 ± 13.3
LVOT, cm	0.6 ± 0.1	0.6 ± 0.1		0.6 ± 0.1	0.7 ± 0.2		1.1 ± 0.1	1.2 ± 0.2
AoR, cm	0.8 ± 0.1	0.9 ± 0.1		0.9 ± 0.2	1.0 ± 0.1		1.5 ± 0.2	1.5 ± 0.3

LVESD, left ventricular end systolic diameter; LVEDD, left ventricular end diastolic diameter; IVS, inter ventricular septum; LVPW, left ventricular posterior wall; LVMI, left ventricular mass index; ESV, end systolic volume; EDV, end diastolic volume; LVOT, left ventricular outflow tract; AoR, aortic root; NS, no significant difference.

[a]$P < .01$ compared with control.

Comparison was not conducted where not applicable due to maturational changes.

strain and strain rate analysis was performed from the DICOM images using vendor-independent clinical echocardiographic software (Image Arena, TomTec Imaging Systems, Munich, Germany). The strain and strain rate values of the apical, mid-cavity, and basal segments of the septal and the lateral walls, as well as global longitudinal strain and strain rate were automatically calculated by the software. An independent investigator (H.J) made all measurements.

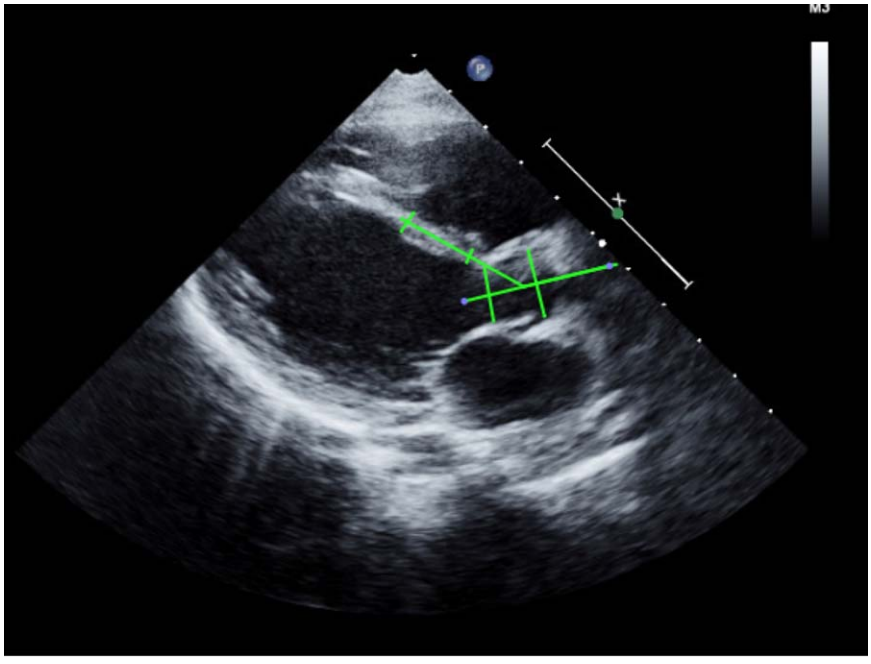

FIGURE 1 Parasternal long-axis view. Demonstrating the method of measuring the angle between the long axis of the aortic root and the midline of the interventricular septum

2.3 | Statistical analysis

All statistical analyses were performed using SPSS for Windows version 21.0 (SPSS, Inc., Chicago, IL). Normally distributed continuous data were expressed as mean ± standard deviation (SD), otherwise, data was presented as median (range). Independent sample *t*-test was used to assess demographic and echocardiographic differences between patients and controls at each follow-up time point and Student paired *t*-test to compare pre- and postoperative data. Relevant non-parametric tests (Mann-Whitney *U* test and Wilcoxon signed-rank test) were used when data was not normally distributed. Linear regression (Pearson's coefficient) was used to determine correlations. Twenty patients' echocardiographic images were randomly selected to evaluate intra- and interobserver agreement using intraclass correlation coefficients and Bland–Altman limit of agreement.[10] To assess intraobserver variability the same observer (H.J) remeasured the AoSA after a month. Interobserver variability, was assessed by comparing the results of two observers (H.J and P.I) blinded to each other. A $P < .05$ was considered as statistically significant.

3 | RESULTS

All patients with CoA included in the study were born at term with male to female ratio of 2, and 19/21 had normal birth weight (>2500 g). Bicuspid AV (BAV) was present in 10/21 (48%) patients and aortic arch hypoplasia in 7/21 (33.3%) patients. All children had surgical correction of the CoA early in life [median age 9 (2–53) days] with 19 receiving end-to-end anastomosis (ETE) and the remaining two had subclavian flap (SF) assessed from the left dorsal thoracotomy. Patients were followed for 8 (2–97) and 850 (208–1508) days after intervention for short and medium-term evaluation, respectively. There were no significant differences between patients and controls regarding age, sex, and body surface area (BSA) (Table 2).

3.1 | Before Intervention

The peak pressure gradient (PG) at the CoA site was 40 (27-121) mm Hg. Three out of 14 patients with available blood pressure (BP) records had systemic hypertension. Mean AoR diameter was not different from normal ($P = .42$), however two patients with BAV had AoR dimensions above 95th percentile for BSA.[11] The AoSA tended to be wider in patients than controls at end diastole, significantly wider at the beginning of systole and considerably wider at peak ejection in the descending aorta (137° ± 6.8° vs. 133° ± 5.6°; $P = .08$, 139° ± 5.9° vs. 135° ± 5.3°; $P = .04$, and 144° ± 6.4° vs. 136° ± 4.1°; $P < .0001$, respectively) (Figure 2). In a multivariate analysis, aortic arch hypoplasia, weight and BAV did not significantly affect the AoSA (end diastole, $P = .22$; beginning of systole, $P = .44$; peak ejection in the descending aorta, $P = .42$).

Mean LV mass (LVM) and mass index (LVMI) were not different from normal ($P = .44$ and $P = .32$, respectively), however two children of 31 and 41 days old had echocardiographic criteria for LV hypertrophy (LVMI 95th percentile for males 80.1 and for females 85.6 g/m$^{2.7}$).[12] Mean LV EF and FS were 55.1% ± 15.9% and 27% ± 10.5%,

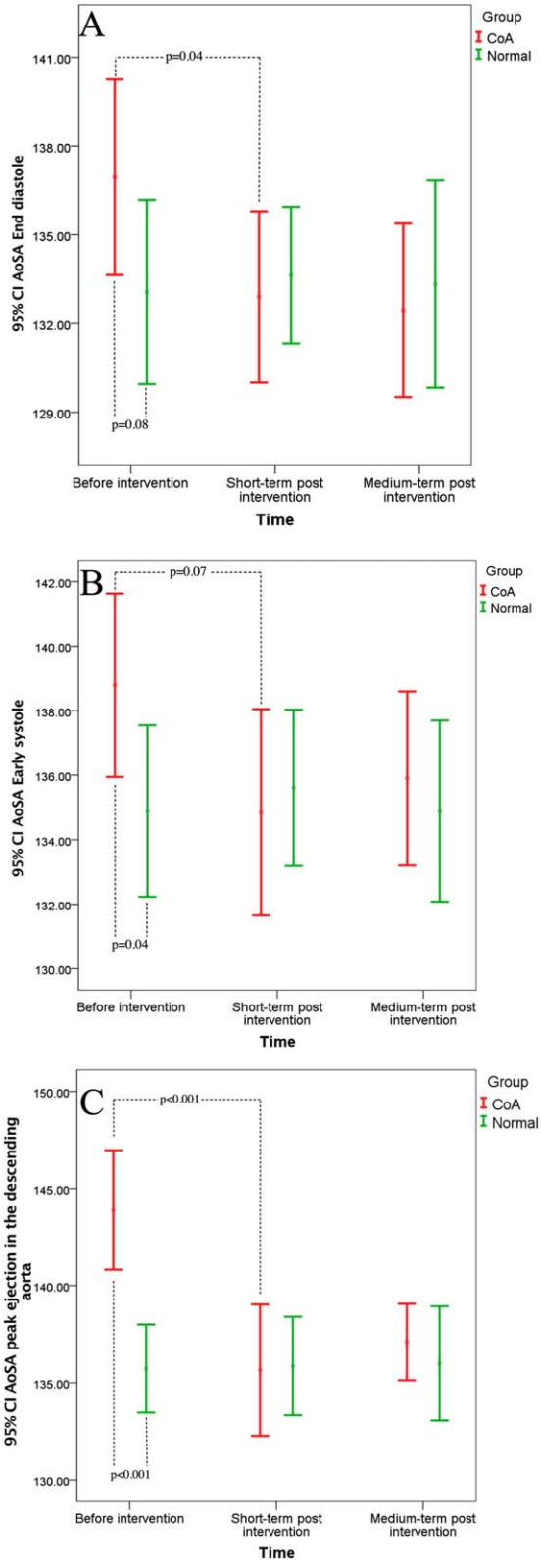

FIGURE 2 Measurements of the AoSA with mean (95% CI) plotted for the three follow-up events. A. AoSA at end diastole; B. AoSA at beginning of systole; C. AoSA at peak ejection flow in the descending aorta

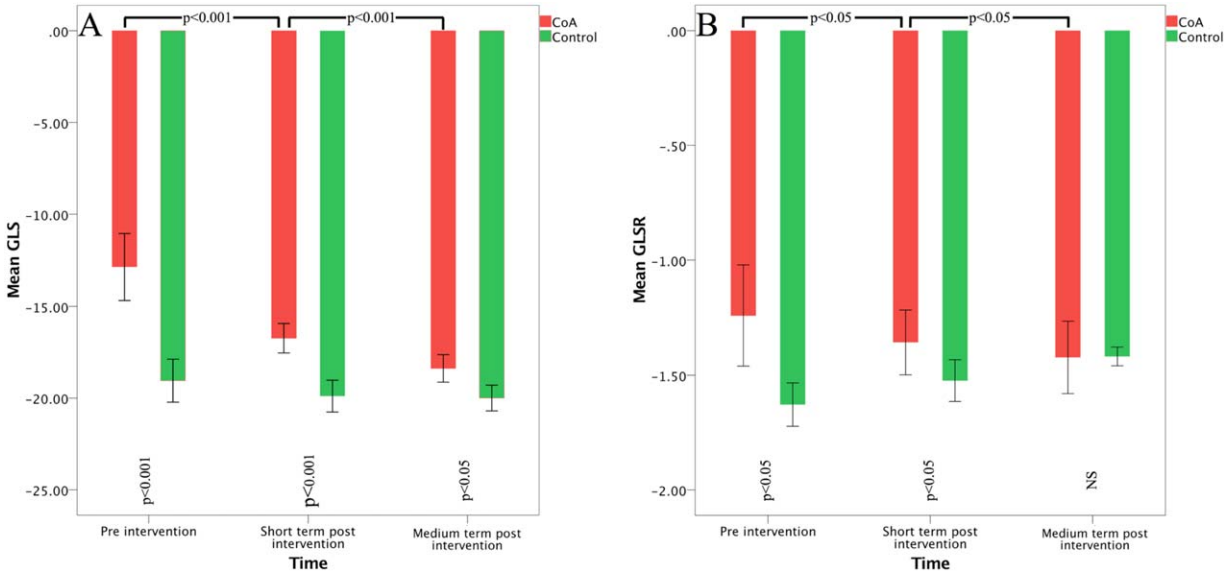

FIGURE 3 Comparison of mean (95% CI) GLS (A) and GLSR (B) for CoA patients and controls in three time intervals

respectively, with nine patients showing signs of cardiogenic pre-shock (EF < 50% and blood pH < 7.3) prior to intervention. LV global longitudinal strain (GLS) and global longitudinal strain rate (GLSR) were both reduced in patients compared with normal (-12.8 ± 3.9 vs. -19.1 ± 3.5; $P < .0001$ and -1.2 ± 0.5 vs. -1.6 ± 0.2; $P < .0001$, respectively) (Figure 3). Basal septal longitudinal strain was reduced and associated with the wide AoSA at end diastole ($R = 0.489$, $P = .034$) and at peak ejection in the descending aorta ($R = 0.522$, $P = .022$), but only tended to do so at the beginning of systole ($R = 0.433$, $P = .064$) (Figure 4).

3.2 | Short term after intervention

After intervention, CoA PG fell from 40 (27-121) to 17 (5-31) mm Hg, $P < .001$. Residual PG (>20 mm Hg) was found in six patients, five of whom had aortic arch hypoplasia before intervention. BP, weight and height were rarely available in the patients' clinical notes. AoSA decreased to normal levels in all three measurements of the cardiac cycle [end diastole (from $137° \pm 6.8°$ to $132° \pm 6.7°$, $P = .04$); beginning of systole (from $139° \pm 5.9°$ to $135° \pm 6.8°$, $P = .07$) and at peak ejection in the descending aorta ($144° \pm 6.4°$ to $136° \pm 7.2°$, $P = .0001$)]. LV EF and FS values normalized. The increase in LV EF correlated with the fall of the AoSA at all three time measurements [end diastole ($R = -0.596$, $P = .009$), beginning of systole ($R = -0.435$, $P = .072$) and at peak ejection in the descending aorta ($R = -0.475$, $P = .046$)]. Insignificantly wide AoSA was found in patients with residual PG (e.g., AoSA at peak ejection in the descending aorta $138.5° \pm 8.9°$ vs. $134.4° \pm 6.3°$, $P = .3$) and aortic arch hypoplasia (e.g., AoSA at peak ejection in the descending aorta $139° \pm 9.2°$ vs. $133.8° \pm 5.5°$, $P = .2$) when compared with the rest of patients. GLS increased ($P < .001$), but remained lower than normal (-16.7 ± 1.7 vs. 19.8 ± 1.9, $P < .0001$). Likewise, GLSR increased ($P = .04$) but yet remained less than normal values (-1.3 ± 0.3 vs. -1.5 ± 0.2, $P = .04$).

3.3 | Medium term after intervention

PG across the repair site remained unchanged 18 (5-38) mm Hg and the same six patients had PG above 20 mm Hg. Systemic hypertension was found in 4/16 patients with available BP measurements, three of them were normotensive before intervention. Mean AoR diameter was not different from normal, except in 3/21 patients with BAV who had values beyond the 95th percentile. AoSA remained not different from normal. The fall of AoSA at peak ejection in the descending aorta correlated with PG prior to intervention ($R = 0.462$, $P = .062$). No differences were found between patients with aortic arch hypoplasia and residual PG when compared with the rest of patients regarding AoSA measured at three time points during the cardiac cycle. Mean LVMI was not different from normal, except in 2/17 patients who had LVMI beyond the 95th percentile for age. Other conventional LV measurements remained unchanged. GLS increased further from short-term ($P < .001$), but remained less than normal values (-18.3 ± 1.7 vs. -20.0 ± 1.6, $P = .002$). GLSR increased ($P = .016$) and became equal to normal values (-1.4 ± 0.3 vs. -1.5 ± 0.1, $P = .6$). The increase in GLS correlated with the fall in AoSA at peak ejection in the descending aorta ($R = 0.534$, $P = .027$).

3.4 | Reproducibility

The intraclass correlation coefficient of the AoSA at peak ejection in the descending aorta showed a good intra- and interobserver agreement [0.86 (95% CI; 0.66-0.95, $P < .0001$ and 0.80 (95% CI; 0.49-0.92, $P = .001$, respectively]. A Bland–Altman plot of the differences plotted against their mean is shown in Figure 5.

4 | DISCUSSION

4.1 | Findings

Children with CoA had abnormally wide AoSA before intervention, particularly at the time of peak ejection in the descending aorta.

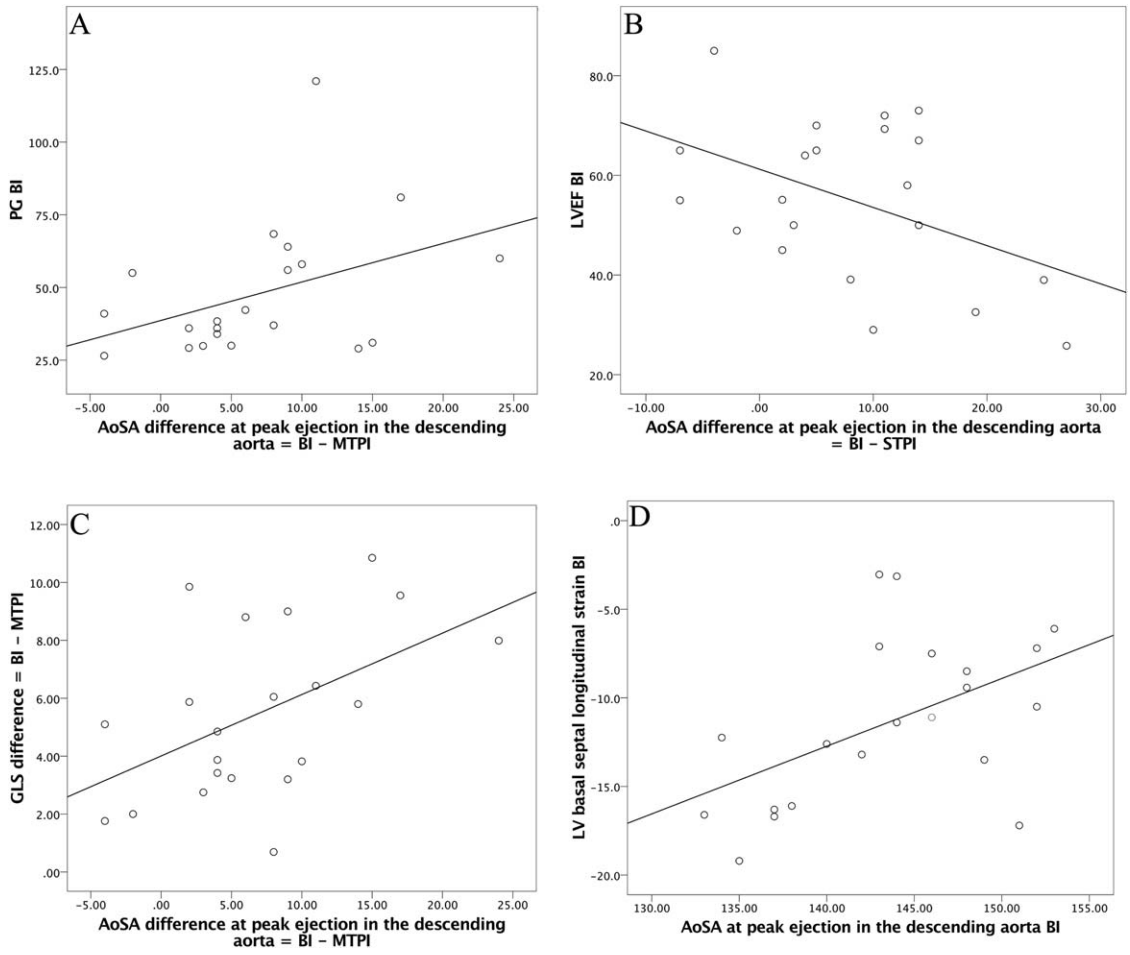

FIGURE 4 Significant correlations between: A. PG BI and AoSA difference (BI-MTPI) at peak ejection in the descending aorta; B. LVEF BI and AoSA difference (BI-STPI) at peak ejection in the descending aorta; C. GLS difference (BI-MTPI) and AoSA difference (BI-MTPI) at peak ejection in the descending aorta; D. LV basal septal longitudinal strain BI and AoSA at peak ejection in the descending aorta BI. PG, pressure gradient; LVEF, left ventricular ejection fraction; BI, before intervention; STPI, short-term postintervention; MTPI, medium-term postintervention

These changes are associated with reduced global LV pump function as well as myocardial deformation in the form of strain and strain rate with the latter correlating with the extent of the angle widening. About half of patients presented in cardiogenic preshock, indicating perhaps the low level of prenatal diagnosis.[13] After correction of the coarctation, significant changes happened with the AoSA and LVEF

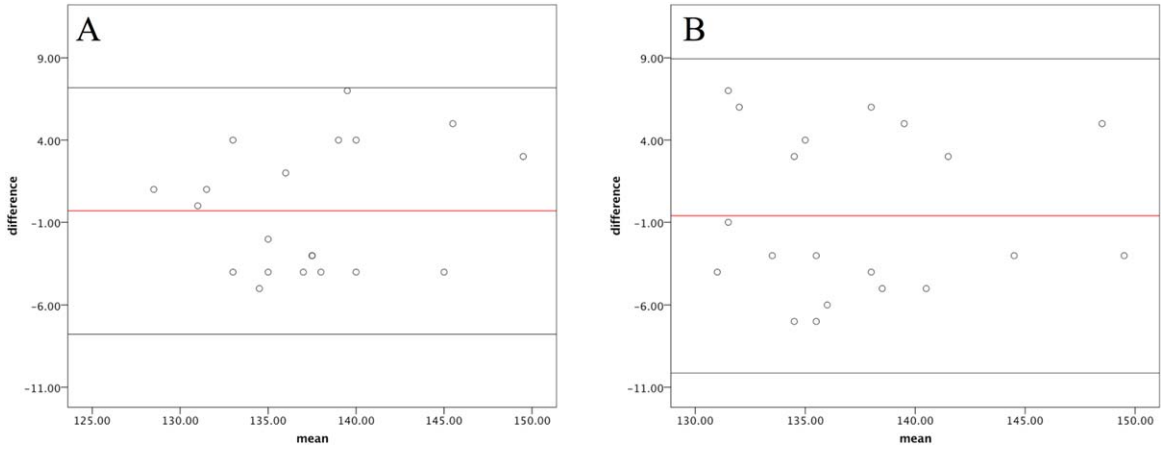

FIGURE 5 Bland–Altman plot of the differences between aortoseptal angulation at peak ejection in descending aorta. A. Intraobserver measurements. B. Interobserver measurements. Red line represents mean and black lines represents mean ± 2 SDs

normalizing and correlating together soon after surgery. Despite the significant improvement in myocardial deformation function after surgery it remained less than normal. Finally, this functional recovery persisted at postoperative mid-term follow-up but with further normalization of myocardial strain, the extent of which correlated with the fall in the AoSA which itself correlated with the severity of the preoperative CoA gradient. Despite all these changes, LV mass was normal preoperatively and remained unchanged.

4.2 | Data interpretation

Pressure afterload is known to have drastic effects on the left ventricle with reactive hypertrophy, increased mass and impaired systolic and diastolic function.[2] The exact impact of pressure afterload on each of these changes differs according to the severity of the condition, its duration and also other related pathologies, for example, systemic hypertension.[14] Our group of children all had CoA as the cause for pressure afterload with only few with additional hypertension. The 48% with bicuspid aortic valve did not have any significant stenosis or significantly different AoSA. Thus, the changes we have reported seem to be related solely to the CoA. Indeed all components of LV function were abnormal, even ejection fraction which is known to be the last to fall, in adults.[15] In addition, segmental deformation of the basal region was significantly reduced being the closest to the level of increased pressure afterload, as we previously showed.[16] Interestingly, such pump function changes were not in isolation but were related to the increased AoSA which itself was related to the severity of the CoA obstruction. This finding suggests an additional function abnormality as a result of the pressure afterload which is the angle between the anatomical LVOT and that of the proximal ascending aorta. The recovery of the angle after correction of the CoA in the presence of normal aortic root diameter suggests that the former is related to the obstruction rather than a potential anomalous root. Since LV dimensions did not change after surgery it is very likely that the fall in the angle is caused by the normalization of the ascending aorta function during ejection with the removal of the resistance of the descending aorta. These findings suggest that the angle widening is caused by the increased pressure in the ascending aorta with its anatomical and functional consequences. Relying on LV mass measurements response to pressure afterload that early in life seem to be of negligible value.

4.3 | Clinical implications

Even in neonates with CoA, the left ventricular function is inherently abnormal very early in life and recovers after removal of the pressure afterload. The anomaly seems also to be associated with deformed geometric relationship between the ascending aorta and the left ventricle, which is corrected after surgery, thus preserving a normal cardiac design. It is also apparent that the latter is determined by the severity of the CoA obstruction, thus suggesting that early removal of the pressure resistance in the descending aorta should increase the chances of a normalized LV-Aortic function, in addition to its known benefit on survival.[17] Furthermore, a potential relation between AoSA after intervention, aortic arch hypoplasia and residual PG are expected to have worse impact on LV dysfunction as a result of the pressure afterload. However, this needs to be confirmed in a larger cohort of patients.

5 | LIMITATIONS

The retrospective nature of the study and the small sample size account for most of the limitations. Incomplete data for some measurements is another limitation. The presence of bicuspid aortic valve with its known aortopathy in 48% of patients might have played a role in the angle widening but this effect is likely to be minimum since the angle normalized after correction of the CoA lesion and without touching the aortic valve. Finally, as the LVOT is dynamic, the echocardiographic measurement of the AoSA at a set point in the cardiac cycle might be considered as an oversimplification of this complex morphology. However, the high level of intra- and interobserver agreement justifies our measures.

6 | CONCLUSION

Aortic-septal angle is abnormally wide in neonates with CoA and correlates with the severity of the obstruction. This is associated with compromised LV global and basal deformation function which all normalize after removal of the pressure afterload. These findings support the early removal of the pressure afterload and the use of these parameters in the management and follow-up of patients with CoA, even in neonates.

CONFLICT OF INTEREST

None.

AUTHOR CONTRIBUTIONS

Haki Jashari: Concept and Design, Data Analysis/Interpretation, Drafting Article, Critical Revision of Article, Statistics

Katarina Lannering: Data Analysis/Interpretation, Critical Revision of Article, Approval of article

Mats Mellander: Data Analysis/Interpretation, Critical Revision of Article, Approval of article

Pranvera Ibrahimi: Data Analysis/Interpretation, Critical Revision of Article, Approval of article

Annika Rydberg: Data Analysis/Interpretation, Critical Revision of Article, Approval of article

Michael Henein: Concept and Design, Data Analysis/Interpretation, Critical Revision of Article, Statistics, Approval of article

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