HEART RHYTHM DISORDERS

Abnormalities of left atrial function after cardioversion: an atrial strain rate study

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Background and objectives: The role of atrial myocardial dysfunction after cardioversion is unclear. In a comparison of patients after successful cardioversion from chronic atrial fibrillation (CAF) and normal controls, we sought to determine whether Doppler-derived atrial strain rate (A-sr) could be used to measure global left atrial function and whether A-sr was reduced in patients with CAF.

Methods: A-sr was measured from the basal septal, lateral, inferior and anterior atrial walls from the apical four-chamber and two-chamber views in 37 patients with CAF who had been cardioverted to sinus rhythm and followed up for 6 months, and in a cohort of 37 healthy people. Conventional measures of atrial function included peak transmitral A-wave velocity, A-wave velocity time integral, atrial fraction and the left atrial ejection fraction. Doppler tissue imaging was used to estimate atrial contraction velocity (A' velocity). In addition to amplitude parameters, the time to peak A-sr was measured from aortic valve closure.

Results: Immediately after cardioversion, A-sr in the CAF cohort (baseline) was significantly lower than in controls (mean (SD) -0.53 (0.31) v -1.6 (0.75) s⁻¹; $p < 0.001$); the A-sr correlated with A' velocity ($r = 0.63$; $p < 0.001$) in patients. Atrial function improved over time, with maximal change observed in the initial 4 weeks after cardioversion. The time to peak A-sr was increased in the CAF group compared with controls (0.55 (0.15) v 0.46 (0.12) s), but this failed to normalise over time.

Conclusion: A-sr is a descriptor of atrial function, which is reduced after cardioversion from CAF and subsequently recovers.

The left atrium serves as a reservoir for blood during ventricular systole, a conduit for passage of blood from the pulmonary veins to the left ventricle and a contractile chamber that augments left ventricular filling. The atria contribute up to 30% of left ventricular filling and cardiac output, so that atrial arrhythmias may have adverse cardiac effects.¹ Atrial fibrillation is the most common arrhythmia, and its incidence increases with age.²

The status of the atrial myocardium in patients with CAF is ill defined. However, new myocardial imaging tools such as tissue Doppler^{3,4} and strain rate imaging^{5,6} may provide a new window to understand atrial contractile function. Such information may be of value in predicting the likelihood of recurrent atrial fibrillation, with obvious implications for the selection of patients for anti-arrhythmic management. Atrial fibrillation is associated with an increase in the incidence of stroke,⁷ as a consequence of thromboembolism originating from within the dysfunctional atrium or the left atrial appendage. Hence, quantification of atrial dysfunction may help identify the subjects in whom anticoagulation is mandatory.

Intraoperative⁸ and catheter-based radiofrequency ablation procedures⁹ are currently used as treatments for atrial fibrillation. In both these instances, A-sr could be used to determine segmental changes in atrial function. Identifying severe segmental atrial dysfunction despite successful restoration of sinus rhythm by either technique could warrant other treatments such as institution of anticoagulation. We therefore sought to determine whether (1) atrial strain rate (A-sr) could determine intrinsic longitudinal atrial contractility; (2) A-sr was decreased compared with controls in a cohort of patients with CAF; (3) A-sr returned to normal in patients with CAF after successful cardioversion and the maintenance of sinus rhythm; (4) A-sr correlated with traditional parameters of atrial function; and (5) to estimate the time to peak A-sr (tA-sr) as a measure of atrial activation.

METHODS

Study design

A total of 37 patients with chronic atrial fibrillation (CAF; 28 men and 9 women) were evaluated at baseline (within 4 h after cardioversion) and at 1 and 6 months after cardioversion in sinus rhythm. They were compared with a cohort of 37 healthy people (10 men and 27 women). None of the controls had a history of ischaemic heart disease or marked valvular abnormalities, peripheral vascular disease, cerebrovascular disease, hypertension or diabetes. None of the controls were receiving cardioactive drugs.

The 37 patients with CAF had atrial fibrillation for a mean duration of 35.8 weeks. All enrolled patients had sinus rhythm restored by electrical cardioversion and had not undergone catheter ablation or the Maze procedure. None of the enrolled patients had greater than mild to moderate valvular regurgitation. Left ventricular function was mildly impaired in the 7 patients who had a history of myocardial infarction, and 13 were hypertensive. At baseline and during the follow-up period of 6 months, 11 patients were taking digoxin, 9 were on sotalol and 11 were on amiodarone. The study was approved by the Committee for Human Research at Westmead Hospital (Sydney, New South Wales, Australia).

Standard transthoracic echocardiography

Doppler M-mode and two-dimensional echocardiography were carried out according to established clinical laboratory practice using commercially available equipment (Vivid 5, General Electric Medical Systems, Horten, Norway) equipped with 3.5 MHz variable-frequency harmonic-phased array transducers.

Abbreviations: A-sr, atrial strain rate; AVC, aortic valve closure; BSA, body surface area; CAF, chronic atrial fibrillation; DTI, Doppler tissue imaging; LAEF, left atrial ejection fraction; LAESV, left atrial end systolic volume; tA-sr, time to peak A-sr; VTI, velocity time integral

Table 1 Demographic and echocardiographic variables of the control and chronic atrial fibrillation cohorts (A) and those adjusted for age and body surface area (B)

	Controls (n = 37)	CAF (n = 37)	p Value
<i>(A) Variables of the control and CAF cohorts</i>			
Age (years)	59.5 (8)	66.4 (10)	0.002
HR (bpm)	68 (10)	64 (16)	NS
Height (cm)	166.5 (8)	172 (10)	0.01
Weight (kg)	73.2 (18)	82.4 (17)	0.03
BSA (m ²)	1.81 (0.23)	1.96 (0.23)	0.006
LA diameter (M-mode)	36.6 (5.1)	48.2 (4.6)	0.001
LAESV (ml)	37.8 (8.2)	76.2 (23.2)	0.001
LAEF (%)	51.7 (7.4)	30.2 (9.7)	0.001
A velocity (m/s)	0.75 (0.2)	0.34 (0.16)	0.001
A VTI (cm)	12.4 (3.5)	5.7 (2.6)	0.001
Atrial fraction (%)	44.8 (10.1)	23.5 (9.3)	0.001
A' velocity (cm/s)	9.4 (1.7)	3.9 (1.1)	0.001
Septal A-sr (s ⁻¹)	-1.6 (0.72)	-0.53 (0.3)	0.001
tA-sr (s)	0.47 (0.11)	0.55 (0.16)	0.02
Peak positive atrial strain	21.1 (8.8)	10.8 (7.6)	0.001
<i>(B) Variables adjusted for age and BSA</i>			
HR (bpm)	69 (2.3)	63 (2.3)	NS
LA diameter (M-mode)	37.4 (1)	48.2 (1)	0.001
LAESV (ml)	37.7 (3.1)	76.2 (3.2)	0.001
LAEF (%)	50.1 (2)	31.8 (2)	0.001
A velocity (m/s)	0.76 (0.03)	0.34 (0.03)	0.001
A VTI (cm)	12.5 (1)	5.7 (1)	0.001
Atrial fraction (%)	44.7 (1.7)	23.5 (1.8)	0.001
A' velocity (cm/s)	9.4 (0.3)	3.8 (0.3)	0.001
Septal A-sr (s ⁻¹)	-1.5 (0.1)	-0.64 (0.1)	0.001
tA-sr (s)	0.47 (0.03)	0.56 (0.03)	0.02
Peak positive atrial strain	21.1 (1.6)	10.4 (1.6)	0.001

A velocity, peak mitral inflow atrial contraction velocity; A', peak atrial contraction velocity using Doppler tissue imaging; A-sr, peak atrial strain rate; BSA, body surface area; CAF, chronic atrial fibrillation; HR, heart rate; LA, left atrium; LAEF, left atrial ejection fraction; LAESV, left atrial end systolic volume (maximal left atrial volume during ventricular systole); tA-sr, time to peak A-sr; VTI, velocity time integral.

The left atrial volume was calculated from the apical four-chamber and apical two-chamber zoomed views, using the biplane method of discs^{10, 11} to obtain left atrial end systolic volume (LAESV, the maximum left atrial volume in ventricular systole) and left atrial end diastolic volume (the minimum volume in ventricular diastole). Left atrial stroke volume was estimated as the difference between the LAESV and the left atrial end diastolic volume. The left atrial ejection fraction (LAEF) was calculated as (left atrial stroke volume/LAESV) × 100.¹¹ The left ventricular ejection fraction was measured in all patients by the biplane method of discs from the apical two-chamber and four-chamber views.¹²

Mitral inflow velocity was obtained by pulsed-wave Doppler sampling at the tips of the mitral leaflets using a sample volume of 5 mm axial length from the apical four-chamber view at a sweep speed of 100 mm/s. The peak velocity of atrial

contraction in late diastole (A-wave velocity) was measured.^{13, 14} The velocity time integral (VTI) of the mitral A wave was measured and the atrial fraction was estimated as A VTI divided by the total VTI of mitral inflow,^{13, 14} and an average of three beats was analysed.

Pulsed-wave Doppler tissue imaging

Peak velocity in late diastole secondary to atrial contraction (A') was measured in all subjects using pulsed-wave Doppler at a sweep speed of 100 mm/s. The pulsed-wave Doppler sample volume was placed on the atrial side of the mitral annulus at the basal interatrial septum in the apical four-chamber view.^{4, 15} Special care was taken to align the Doppler beam to the interatrial septum to optimise Doppler measurements obtained during end expiration. An average of three beats was measured.

Table 2 Strain rate measured from the basal atrial segments in the chronic atrial fibrillation and control groups at baseline

	Controls (n = 37)	CAF (n = 37)	p Value
Basal septal E-sr (s ⁻¹)	-0.99 (0.51)	-0.63 (0.39)	0.001
Basal septal A-sr (s ⁻¹)	-1.6 (0.72)	-0.53 (0.31)	0.001
Septal tA-sr (s)	0.47 (0.11)	0.55 (0.16)	0.017
Basal lateral E-sr (s ⁻¹)	-1.3 (0.62)	-1.2 (0.78)	NS
Basal lateral A-sr (s ⁻¹)	-0.95 (0.64)	-0.34 (0.25)	0.001
Basal inferior E-sr (s ⁻¹)	-1.5 (0.84)	-1.1 (0.91)	NS
Basal inferior A-sr (s ⁻¹)	-1.8 (0.96)	-0.6 (0.38)	0.001
Inferior tA-sr (s)	0.47 (0.13)	0.56 (0.13)	0.005
Basal anterior E-sr (s ⁻¹)	-0.64 (0.4)	-0.48 (0.3)	0.01
Basal anterior A-sr (s ⁻¹)	-0.90 (0.6)	-0.63 (0.36)	0.01

A-sr, peak atrial strain rate in late diastole; CAF, chronic atrial fibrillation; E-sr, peak strain rate in early diastole; tA-sr, duration from aortic valve closure to peak atrial strain rate.

Atrial strain and strain rate imaging

Images were obtained using a narrow sector (frame rate >110 fps), and attempts were made to align the atrial wall parallel to the Doppler beam. Because of the thin atrial walls, a narrow (10×2 mm) sample volume was selected and placed in the basal septal, inferior, lateral and anterior walls of the atrium from the apical four-chamber and two-chamber views. The sample volume was placed in the middle of the respective basal segment and the image was tracked frame by frame, ensuring in each frame that the sample volume was moved to its original location in the middle of the segment using dedicated software available on an offline measuring station (EchoPac PC, GE-Vingmed, Horten, Norway). The peak strain rate in early diastole and that in late diastole (A-sr) corresponding to atrial contraction were measured (fig 1). Gaussian smoothing (60) was applied before peak strain rate was measured. For each subject, measurements were obtained as an average of two consecutive cycles. If the angle of interrogation exceeded 30°, the patient was excluded from final analysis. The four basal atrial segments (septal, lateral, inferior and anterior) were analysed at baseline. However, in several subjects, the angle of interrogation for the basal anterior and basal lateral segments exceeded 30°. For the serial follow-up, A-sr was studied only in the basal septal and basal inferior walls.

In addition to amplitude parameters, the timing parameter of the time from aortic valve closure (AVC) to tA-sr was measured. The AVC was obtained from Doppler tissue images as the point where the descent of the S velocity intersected the zero line. The tA-sr was measured as the duration from AVC to the peak A-sr.

Observer agreement

In 10 randomly selected studies from each cohort, two readers independently estimated the peak A' velocity, the A-sr, the peak A velocity, and the A VTI and atrial fraction. One observer evaluated the same 10 studies on a separate occasion to determine the intraobserver variability.

Analysis

All values are expressed as mean (standard deviation (SD)) unless otherwise stated. Differences between groups were examined by two-sample Student's *t* test. Repeated-measures analysis of variance was carried out to estimate within-patient changes for the group enrolled in long-term follow-up. Spearman's rank correlation was used to examine the pairwise associations between A-sr and LAEF, peak A velocity, A wave VTI, atrial fraction and A' velocity. Bland-Altman analysis¹⁶ was used to assess reproducibility within and between observers. Data were analysed using Statview Student V.4.0 and SPSS for Windows V.12.0.

RESULTS

Clinical and echocardiographic characteristics

Table 1A summarises the demographic and echocardiographic variables of the normal and CAF cohorts. As shown in table 1A, a small yet significant difference in age was observed between the two groups, with the normal cohort being younger than the CAF cohort. Additionally, the body surface area (BSA) was also significantly higher in the CAF group. Traditional parameters of atrial function—namely, the A velocity, A wave VTI, atrial fraction and LAEF—were significantly higher in the controls than those in the atrial fibrillation group at baseline (table 1A). Doppler-derived A-sr and peak positive atrial strain were also lower in the CAF group, and the tA-sr was prolonged.

As atrial size and function are thought to be altered with ageing and also with varying BSA, a separate statistical analysis

was carried out to compare the CAF and control groups, adjusting for age and BSA as covariates. However, significant differences between the two groups in left atrial size (LAESV) and in parameters of atrial function (including LAEF, peak A velocity, A wave VTI, atrial fraction, A' velocity and A-sr) remained even after correcting for the effects of age and BSA (table 1B).

Correlation of A-sr to traditional parameters of atrial function

A-sr showed a moderate correlation to traditional parameters of atrial function, including the peak A velocity (Spearman's $r = 0.56$; $p = 0.001$), the A wave VTI (Spearman's $r = 0.54$; $p = 0.001$) and the atrial fraction (Spearman's $\rho = 0.53$; $p = 0.001$; fig 1A–C). The A-sr also correlated with the LAEF (Spearman's $r = 0.65$; $p = 0.001$) and the A' velocity obtained from Doppler tissue imaging (DTI) measurements (Spearman's $r = 0.63$; $p = 0.001$; fig 1D,E).

A-sr in atrial fibrillation and controls

No significant differences were noted between the contractility of the basal septal and basal inferior segments in subjects in each group at baseline immediately after cardioversion (table 2). In patients, velocities in the basal lateral and anterior walls were lower than those noted in the basal septal and inferior walls. A paired *t* test showed no significant difference between the basal septal and basal inferior segments (mean (SD) septal A-sr: -1.6 (0.72) ν -1.8 (0.96) s^{-1} ; $p = 0.72$) and between the basal lateral and basal anterior segments (-0.95 (0.64) ν -0.9 (0.6) s^{-1} ; $p = 0.8$). Both the basal lateral and basal anterior A-sr were significantly lower than the basal septal A-sr ($p = 0.01$ and 0.08 , respectively). This may relate to the angle of interrogation in these segments. A significantly lower A-sr was noted in the CAF group than in the controls.

The tA-sr was significantly longer in the CAF group than in the normal cohort at baseline. A significant difference was noted in the tA-sr between the groups even after adjusting for heart rate (controls ν CAF: mean (SD) 0.49 (0.19) ν 0.54 (0.19) s). There was no significant correlation noted between left atrial size and the tA-sr.

Temporal improvement in atrial function with maintenance of sinus rhythm

An increase in A-sr was noted in the atrial fibrillation cohort when followed up temporally after cardioversion (table 3). The maximum within-patient improvement in A-sr was noted in the first 4 weeks after cardioversion, similar to other parameters of atrial function. Interestingly, although the amplitude parameters showed a temporal improvement, timing parameters of tA-sr remained unaltered in the CAF cohort.

Observer variability

Ten subjects in each group were randomly selected for interobserver and intraobserver variability analysis. Peak strain rate in early diastole and A-sr were remeasured by the same observer and by a second independent observer from the digital data. Bland-Altman analysis for these parameters showed no evidence of any systematic difference between the two sets of measurements. Table 4 shows the mean difference and confidence intervals of interobserver and intraobserver variabilities.

DISCUSSION

We have shown that A-sr is a feasible and reproducible marker of longitudinal atrial contractility. The A-sr was reduced in the CAF group, but showed a significant increase in magnitude,

Figure 1 (A) Correlation of septal atrial strain rate (A-sr) to peak (Pk) A velocity (VEL). (B) Correlation of septal A-sr to A wave velocity time integral (VTI). (C) Correlation of septal A-sr to atrial fraction. (D) Correlation of septal A-sr to left atrial ejection fraction (LAEF). (E) Correlation of septal A-sr to A' velocity (Doppler tissue imaging).

with maintenance of sinus rhythm. This recovery of the A-sr parallels that noted in traditional parameters of atrial function. Nonetheless, the persisting abnormality of tA-sr suggests ongoing atrial dysfunction in this group.

Atrial tissue velocity versus strain rate parameters

DTI can evaluate regional myocardial function.^{3,17} We previously showed that DTI can be used to evaluate global and segmental atrial functions.⁴ However, a major limitation of DTI

Table 3 Time course for improvement in atrial function parameters after the restoration of sinus rhythm in patients with chronic atrial fibrillation

Parameter	Immediately after CV	1 month after CV	6 months after CV	p Value
Basal septal A-sr (s ⁻¹)	-0.53 (0.31)	-1.01 (0.49)	-1.1 (0.49)	0.001
A velocity (m/s)	0.34 (0.16)	0.71 (0.25)	0.77 (0.28)	0.001
A wave VTI (cm)	5.7 (2.6)	10.4 (4.3)	10.7 (3.3)	0.001
Atrial fraction (%)	24 (9)	36.4 (11)	37.8 (10)	0.001
LAEF (%)	30.2 (10)	36 (11)	42 (13)	0.001
A' velocity (cm/s)	3.9 (1.1)	7.3 (1.9)	8.5 (2.2)	0.001
tA-sr (s)	0.55 (0.16)	0.58 (0.16)	0.59 (0.17)	NS

A velocity, peak mitral inflow atrial contraction velocity; A', peak atrial contraction velocity using Doppler tissue imaging; A-sr, atrial strain rate imaging; CV, cardioversion; LAEF, left atrial ejection fraction; tA-sr, duration from aortic valve closure to peak atrial strain rate; VTI, velocity time integral.

is that it cannot distinguish between intrinsic myocardial motion and that produced by passive translatory motion due to the effects of tethering. More recently, DTI-derived strain and strain rate imaging have been evaluated in ventricular myocardium.⁵⁻⁶ Strain and strain rate imaging of the ventricular myocardium have been extensively reported in normal¹⁸⁻¹⁹ and in diseased states.²⁰⁻²² However, there is a paucity of data on strain and strain rate imaging of the atria.

The difference in atrial and ventricular emptying and filling occurs in a complementary fashion. Thus, in systole the ventricle contracts while the atrium fills, and conversely in diastole the ventricle fills while the atrium undergoes passive and active emptying. We would therefore assume that opposing directions of longitudinal contraction should be obtained from atrial and ventricular sampling. However, with DTI, concordant motion of the atrium to that of the ventricle is observed, presumably reflecting the inability of tissue Doppler imaging to distinguish atrial contraction from mitral annular and ventricular motion. By contrast, the longitudinal shortening and lengthening of the atrium are discordant with ventricular longitudinal motion, because the atrium fills during ventricular systole and empties during ventricular diastole (fig 2). The recognition of this discordance is possible because A-sr, unlike DTI-derived A' velocity, shows a site-specific directional difference.

A-sr as a marker of atrial function

Evidence shows atrial dysfunction and stunning occur immediately after cardioversion. Both invasive²³⁻²⁴ and non-invasive¹³⁻²⁵⁻²⁶ techniques have evaluated the recovery of atrial function, with restoration of sinus rhythm. These studies showed a dissociation between electrical and mechanical

recovery after successful cardioversion to sinus rhythm. They also showed a temporal improvement in atrial function in the first 3-4 weeks after cardioversion.¹³⁻²⁶ Similar to traditional parameters, such as the peak A velocity and A VTI, A-sr shows reduced atrial contractility immediately after cardioversion. A significant temporal improvement was noted, as has been described previously. The main improvement in atrial function occurs within the first 4 weeks after sinus rhythm is restored.

The peak A velocity has previously been shown to normalise 4 weeks after cardioversion.¹³⁻²⁶ However, this study of A-sr shows a persisting degree of atrial dysfunction despite the maintenance of sinus rhythm for a 6-month period. This dysfunction could warrant the longer-term use of anti-arrhythmic treatment in the CAF cohort. Likewise, similar studies of the A-sr in paroxysmal atrial fibrillation may show differences in the A-sr compared with the CAF group.

Timing of atrial contraction

Delayed atrial contraction was also observed in the patients with CAF at baseline after cardioversion. However, with temporal follow-up over 6 months, no decrease was observed in the delayed atrial activation, suggesting that atrial myocardium is perhaps intrinsically altered in atrial fibrillation. Thus, although contractile function is restored in some measure, atrial conduction remains prolonged. This difference in atrial myocardium could explain the increased propensity for these patients to develop subsequent atrial fibrillation, and perhaps may represent an atrial myopathic process that persists despite the restoration of sinus rhythm. An alternative explanation is that the left atrial size is considerably larger in the CAF group and this may account for the prolongation in the tA-sr. However, in this preliminary cohort, no correlation was observed between atrial size and tA-sr.

Limitations

The reference standards used for comparison were traditional echocardiographic parameters. Invasive tests such as cardiac catheterisation to evaluate left atrial pressure were not considered feasible. Finally, the A-sr is Doppler derived and remains angle dependent, making measurements from the basal anterior and lateral walls difficult.

Conclusion

A-sr is a site-specific measure of intrinsic atrial contractility that is reduced in CAF after cardioversion to sinus rhythm. A temporal improvement is noted in the A-sr, with maintenance of sinus rhythm. However, atrial contraction is delayed in the CAF cohort and the tA-sr showed no marked improvement over time. Our data also show that A-sr occurs with differences in regional deformation to left ventricular strain, suggesting that this parameter is a more accurate measure of atrial contractility.

Table 4 Bland-Altman analysis for interobserver and intraobserver variabilities and confidence intervals for A-sr

Parameter	Interobserver mean difference	Interobserver 95% CI	Intraobserver mean difference	Intraobserver 95% CI
Septal peak E-sr (s ⁻¹)	0.385	-0.12 to 0.19	-0.19	-0.22 to 0.19
Septal peak A-sr (s ⁻¹)	-0.137	-0.32 to -0.05	-0.56	-0.17 to 0.06
Septal tA-sr (s)	3.7	-13.6 to 24.5	3.5	-14.9 to 21.9
Lateral peak E-sr (s ⁻¹)	-0.35	-0.63 to -0.08	-0.23	-0.65 to 0.18
Lateral peak A-sr (s ⁻¹)	-0.055	-0.18 to -0.07	-0.106	-0.33 to 0.12
Inferior peak E-sr (s ⁻¹)	-0.04	-0.33 to -0.25	0.19	-0.19 to 0.58
Inferior peak A-sr (s ⁻¹)	-0.15	-0.48 to 0.19	0.31	-0.52 to 1.13
Anterior peak E-sr (s ⁻¹)	0.41	-0.11 to 0.17	0.23	-0.22 to 0.16
Anterior peak A-sr (s ⁻¹)	-0.123	-0.13 to 0.22	-0.11	-0.12 to 0.22

A-sr, atrial strain rate; E-sr, peak strain rate in early diastole.

Figure 2 (A) Atrial strain rate trace obtained with sample volume placed in the basal atrial septum. The vertical red line denotes aortic valve closure (AVC). The horizontal arrow denotes the time from AVC to peak atrial strain rate (tA-sr). (B) Ventricular strain rate trace obtained with sample volume placed in the basal ventricular septum.

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Competing interests: None.

Ethical approval for this study was obtained from the Committee for Human Research, Westmead Hospital. The approval number for this study is HREC99/9/4.11(888). All enrolled patients provided written consent for willingness to participate in this research.

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The contents of this article have not been published as a part or whole in any other journal previously and at present is not under consideration in any other journal for publication.

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Interrupted aortic arch and aortopulmonary window demonstrated on 64-slice multidetector computed tomography angiography

A 3-day-old infant was referred for cardiac assessment because of multiple congenital anomalies including facial dysmorphism, cleft lip and palate and exomphalos minor. Echocardiography revealed type B interrupted aortic arch and large aortopulmonary window. The interventricular septum was intact.

A 64-slice multidetector computed tomography (MSCT) angiography (Sensation 64, Siemens, Germany) was performed using high resolution protocol (slice thickness 0.75 mm, pitch factor 1.2) without electrocardiographic gating. A total of 6 ml of intravenous contrast was injected through a 24 G cannula in the right saphenous vein. No sedation was given and the actual scan time was 1.97 s. Following acquisition, three-dimensional volume rendered images were reconstructed using Syngo InSpace4D application software.

The study illustrated interruption of the aortic arch between the left common carotid and left subclavian arteries, giving rise to the classic "V" sign appearance. The descending aorta was fed by a large patent ductus arteriosus (panel A: BC, brachiocephalic artery; LCC, left common carotid artery; LSC, left subclavian artery; PDA, patent ductus arteriosus). The spatial relationship between the vessels was clearly demonstrated. In addition, there was a large communication existing between the ascending aorta and the proximal main pulmonary artery which represented a large aortopulmonary window (panel B: arrow; AAo, ascending aorta; MPA, main pulmonary artery). Conservative management was advocated subsequently after the diagnosis of trisomy 13 was revealed on karyotyping.

MSCT provided a fast and non-invasive way for accurate morphological evaluation in this case.

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