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Cardiovascular changes after transcatheter endovascular stenting of adult aortic coarctation

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ABSTRACT

Background: Longer term data on efficacy and clinical endpoints relating to transcatheter endovascular stenting in adults with aortic coarctation remains limited. We hypothesised that stenting would have effects on blood pressure, presence and extent of collaterals, left ventricular (LV) mass and vascular function.
Methods: Eighteen patients mean age 31.6 ± 12.8 years were studied with clinical assessment and cardiovascular magnetic resonance before and after (10.2 ± 2.2 months) aortic coarctation endovascular stenting. Fredriksen coarctation index increased and using this index no patient had significant coarctation (index <0.25) after stenting.
Results: Blood pressure decreased (153 ± 17/82 ± 14 *versus* 130 ± 21/69 ± 13 mm Hg; $p<0.001$) unrelated to change in existing anti-hypertensive therapy. LV ejection fraction increased (70 ± 10 *versus* 74 ± 8%; $p=0.01$) and LV mass index decreased (91 ± 24 *versus* 82 ± 20g/m^2; $p=0.003$). Collaterals appeared smaller and the degree of flow through collateral arteries decreased (40 ± 29 *versus* −1 ± 33%; $p<0.001$). Distensibility of the ascending aorta increased (4.0 ± 2.5 *versus* 5.6 ± 3.5 × 10^{-3} mm Hg^{-1}; $p=0.04$). Unexpectedly, right ventricular mass index decreased (35 ± 7 *versus* 30 ± 10g/m^2; $p=0.01$).
Conclusion: All patients underwent successful relief of coarctation by endovascular stenting. Both cardiac and vascular beneficial outcomes were demonstrated. The reduction in LV mass suggests a potential for reduction in risk of adverse events and warrants further study.

1. Introduction

Untreated, aortic coarctation has a poor prognosis, with most patients dying from stroke, coronary heart disease or sudden death, by the fourth decade of life [1].

Coarctation is normally detected in childhood and repaired surgically but it occasionally recurs or presents de novo in adolescence or adulthood when it is characterised by upper body systemic hypertension and adverse cardiovascular remodelling including increased arterial stiffness and left ventricular (LV) hypertrophy.

Although surgical treatment has had a major impact on outcome [2], recently many older patients with coarctation or recoarctation have been treated endovascularly by balloon dilatation and or stent implantation, as a less invasive alternative to surgery. Satisfactory outcomes following endovascular stenting, with respect to procedural risks and relief of gradient, have been reported [3–5]. Additionally, favourable outcomes in early to mid-term follow-up are now reported with respect to blood pressure [5–12] though it has also been documented that arterial hypertension may persist in some patients [13]. Nevertheless, few data exist on the impact of this local approach on both structural and functional cardiovascular abnormalities, which are likely to be the main determinants of long-term cardiovascular risk. Therefore, we aimed to precisely assess prospectively, with cardiovascular magnetic resonance (CMR), the effect of endovascular stenting of aortic coarctation on systemic arterial blood pressure, collateral flow, ascending aortic distensibility and ventricular function and mass.

2. Methods

Eighteen consecutive patients referred for endovascular stenting of coarctation were prospectively studied. The study protocol complies with the Declaration of Helsinki, was approved by the local research ethics committee and all patients gave written, informed consent. Drug therapy prior to, and during the period of follow-up, was recorded and wherever possible no change in anti-hypertensive medication was made over the study period in order to assess the effect of stenting in isolation. No patients required exclusion due to inability to cooperate with CMR.

Abbreviations: LV, left ventricle; RV, right ventricle; CMR, cardiovascular magnetic resonance; CE-CMR, contrast enhanced magnetic resonance angiography; ANOVA, analysis of variance; LVMi, LV mass index; LVEF, LV ejection fraction; LVEDVi, LV end-diastolic volume index; LVESVi, LV end-systolic volume index; RVMi, RV mass index; RVEDVi, RV end-diastolic volume index; RVESVi, RV end-systolic volume index.

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2.1. CMR clinical protocol — anatomy, flow and function

A 1.5 T Siemens Sonata system was used (Siemens AG, Erlangen, Germany). Steady state free precession (SSFP) cines were aligned with the aorta at the site of the coarctation in two planes to assess the narrowest point and demonstrate anatomy. The narrowest diameter of the coarctation and the diameter of the aorta at the level of the diaphragm were measured directly. Complementary data from different sequences, SSFP, turbo spin echo (TSE) and raw data from contrast-enhanced CMR (CE-CMR) angiography were used to determine minimal diameters. All these sequences were used to assess minimal diameters at both baseline and follow-up in planes aligned to demonstrate the coarctation or stent. We have previously shown only small differences between sequences with good correlation of non-invasive measurements with subsequent X-ray angiographic data [14]. The requirement for the different sequences is that they have different relevant pitfalls or artefacts so that in some patients a uniform sequence might not be reliable at both time points, as discussed under limitations. For example, baseline SSFP cines gave reliable measurements of even severe, tight coarctation as did pre processed contrast angiography but in these patients the baseline TSE gave signal dropout in the region of interest. Yet, at follow-up in all patients turbo spin echo gives the clearer delineation of the post-stented aorta, though artefact for the other sequences was not so severe as to render them useless. The anatomical severity of the coarctation was estimated using the Fredriksen coarctation index [15]. In general an index of <0.25 is regarded as a significant obstruction.

$$\textit{Fredriksen Coarctation index} = \frac{(\textit{Minimal Diameter Coarctation})^2}{(\textit{Diaphragmatic Level Aortic Diameter})^2}$$

Percentage coarctation and percentage change in minimal diameter were also estimated using the formulae below.

$$\%\ \textit{Coarctation} = \frac{(100 \times (1 - \textit{Minimal Diameter}))}{\textit{Diaphragmatic Diameter}}$$

SSFP cines acquired included a short axis view of the aortic valve. A short axis stack of eight to eleven 7 mm slices 3 mm apart to cover the entire heart was acquired and measurements of ventricular volumes, function and mass (CMRtools, Imperial College, London, UK) performed by a single observer (SVB-N) using standard methods [16–18]. Measurements were indexed to body surface area. Through-plane, breath-hold, phase contrast velocity mapping for measurement of peak velocity was located using two previous cine acquisitions of the coarctation in perpendicular planes and an in-plane velocity map aligned with the jet at the area of highest velocity. The highest recorded velocity was taken as the peak. Non-breath-hold (NBH), phase contrast, retrospectively gated velocity mapping for quantification of flow was acquired in a proximal and distal plane in the locations prescribed in Fig. 1. NBH is more suitable for volume flow measurement at diaphragm. It is less prone to non-velocity-related phase shifts when acquired outside the central field. As we did not want to mix sequences for comparisons of flow, the proximal velocity map was acquired with the same sequence. The proximal plane was located immediately distal to the coarctation avoiding the region of high velocity (shear) but not too far downstream as this would have included collaterals re-entering. In the case of post stent studies, this alignment was similar but sufficiently distal to the stent itself to avoid artefact. The second acquisition was in a plane transecting the distal descending aorta at the level of the diaphragm for estimation of collateral flow [19–21] (Fig. 1) using the formula:

$$\%\ \textit{collateral flow} = \frac{(\textit{Flow at the diaphragm} - \textit{Flow under the CoA / Stent}) \times 100}{\textit{Flow at the diaphragm}}$$

2.2. Contrast-enhanced magnetic resonance angiography (CE-CMR)

CMR was performed using 3D ultra fast gradient-echo (FLASH) sequences with RF (radiofrequency) spoiling and a phased array surface coil. A breath-hold 3D CE-CMR was acquired in oblique sagittal orientation. Typical parameters used were echo time 1.04 ms; repetition time 2.6 ms; flip angle 25°; matrix size 512×256; slice thickness 1.5 mm; receiver bandwidth 700 Hz per pixel; field of view 30–40 cm. Average acquisition time was 19 s with one signal average. The imaging slab of 60 to 80 mm was partitioned into 40 segments. K-space filling was sequential and the peak gadolinium concentration in the thoracic aorta timed to coincide with sampling at the centre of k-space. No patients required sedation. Gadolinium-DTPA (Magnevist; Schering, Germany, 0.2 mmol/kg) was given using a power injector pump at 2 ml/s. An initial test bolus was given to determine the time to peak contrast concentration. Images were analysed and processed by a single blinded experienced investigator (RHM) using 3D reformatting, multiplanar reformation and maximum intensity projection techniques. Processing typically took less than 15 min. Collaterals were graded (RHM) according to the CE-CMR appearance and blind to clinical data. Number, size and tortuosity contributed to a global qualitative impression and the semi-quantitative 3-point scale grading of appearance as mild, moderate or extensive.

2.3. Measurement and analysis of distensibility

A high resolution small field of view SSFP cine sequence (field of view read 220 mm/voxel size 1.4×1.7×6 mm/~30ms temporal resolution) of the ascending aorta at the level of the right pulmonary artery was acquired. Planimetry of the inner wall was performed by a single observer (SVB) in all frames and the maximum (systolic) and minimum (diastolic) area recorded. Right arm cuff blood pressure measurement was made whilst the patient was lying down within the scanner at the time of the sequence acquisition using CMR compatible equipment (3150 MRI Physiological Monitor-Magnitude; Invivo research, Inc.). Calculation of ascending aortic distensibility was then made using the formula:

$$D = \frac{(A_{\text{syst}} - A_{\text{diast}})}{(A_{diast} \times \textit{Pulse Pressure})}$$

whereby D = Distensibility mm Hg^{-1}, A_{syst} = maximum aortic area in systole (mm^2), A_{diast} = minimum aortic area in diastole (mm^2), pulse pressure = systolic–diastolic right arm systemic blood pressure during scan [22].

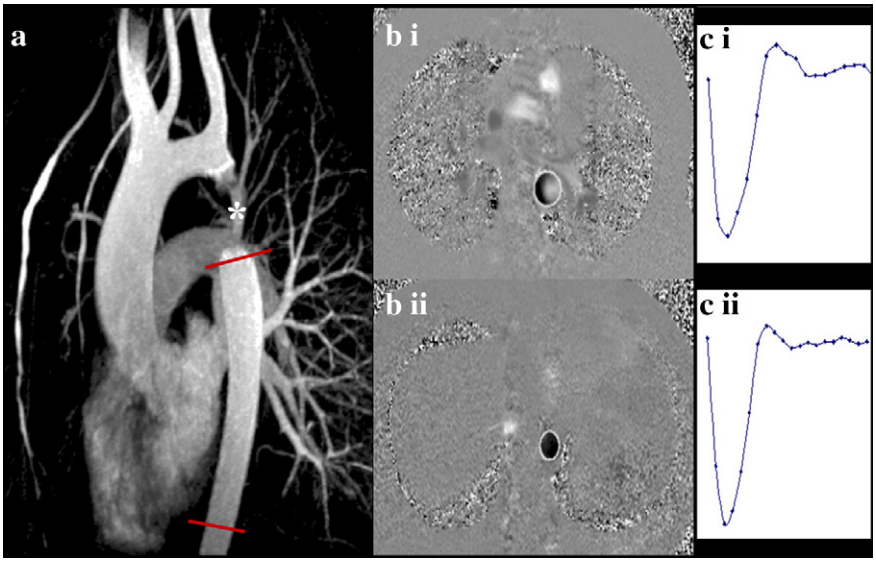

Fig. 1. Quantifying collateral flow with CMR. a) The site of acquisition for measurements of collateral flow is shown by the red lines in this example of a post-endovascular stent patient. b) Resulting phase encoded velocity maps from the proximal acquisition (above; b i) and the distal acquisition (below; b ii) are shown. The stent artefact on CE-CMR may be noted (asterisk). Planimetry of the regions of interest was performed. c) The resultant flow curves plotted for flow volume calculation are shown. Flow was expressed in ml/s, proximally at a site immediately after the coarctation or stent (c i) and in the distal aorta at the level of the diaphragm (c ii). The difference in flow was documented and expressed as a percentage of diaphragmatic flow volume according to the formula $(F_{\text{dist}} - F_{\text{prox}})/F_{\text{dist}} \times 100\%$ whereby F = Flow in ml/s, dist = distal (at the level of the diaphragm), prox = proximal (distal to the level of the coarctation).