International Journal of Cardiology 119 (2007) 21–27
International Journal of Cardiology
www.elsevier.com/locate/ijcard

Impact of transverse aortic arch hypoplasia after surgical repair of aortic coarctation: An exercise echo and magnetic resonance imaging study

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Received 8 February 2006; received in revised form 3 June 2006; accepted 8 July 2006
Available online 17 October 2006

Abstract

Background: We sought to assess the impact of persistent hypoplasia of the transverse aortic arch (TAA) after repair of aortic coarctation (AoC), on blood pressure response to exercise, left ventricular (LV) hypertrophy and presence of collateral circulation.
Methods: 34 consecutive patients with end-to-end repair of AoC (age at repair 3.2±2.5 years) underwent exercise echocardiography and magnetic resonance imaging (MRI) at 24±7 years of age (range 11.3 to 44.6 years). Systolic Doppler pressure gradient (SPG) across the descending aorta and blood pressure at the right arm were measured at baseline and every minute throughout all exercise. Magnetic resonance imaging was used to measure LV mass index, presence and amount of collateral flow, and the diameters of the aortic isthmus and TAA indexed to the diameter of the diaphragmatic.
Results: Aortic isthmus index was higher than that of the TAA ($p=0.006$). We observed LV hypertrophy in 15 patients (45%) and presence of collateral circulation in 14 (41%). Eighteen patients (53%) had an abnormal blood pressure response to exercise. Patients with abnormal pressure response to exercise had smaller TAA index ($p=0.0005$), but similar aortic isthmus index ($p=0.09$). They also had higher exercise SPG ($p<0.0001$), higher LV mass index ($p<0.0001$) and prevalence of LV hypertrophy ($p=0.007$), higher prevalence of collateral circulation ($p<0.0001$) and a higher amount of collateral flow ($p<0.0001$). TAA index, but not aortic isthmus index, correlated with exercise blood pressure ($r=-0.59$, $p=0.003$), exercise SPG ($r=-0.70$, $p=0.0005$), amount of collateral flow ($r=-0.74$, $p=0.0002$) and LV mass index ($r=-0.68$, $p=0.0007$).
Conclusions: After repair of AoC, hypoplasia of the TAA may be responsible for abnormal blood pressure response to exercise, persistence of collateral circulation and LV hypertrophy.

Keywords: Aorta; Coarctation; Hypertrophy; Hypertension; Echocardiography

1. Introduction

Patients operated for coarctation of the aorta (AoC) may present at follow-up increased blood pressure at rest or during exercise [1], leading to left ventricular (LV) hypertrophy and increased mortality when compared to the general population [2,3].

Different potential mechanisms have been proposed to explain arterial hypertension in the absence of recoarctation, like resetting of the cardiopulmonary baroreflex with excessive LV function [4], abnormal aortic arch geometry [5] and upper body conduit artery dysfunction [6–8]. We hypothesized that hypoplasia of the transverse aortic arch (TAA) may cause a relevant obstruction during exercise-induced increase of cardiac output [8], and that this obstruction would lead to abnormal blood pressure response to exercise, LV hypertrophy and persistence of collateral circulation.

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doi:10.1016/j.ijcard.2006.07.036

2. Materials and methods

In a prospective fashion, all patients with repaired AoC who accomplished inclusion criteria and who were attending our outpatient clinic between September 2003 and August 2005 were invited to take part in the study. Study protocol consisted of a magnetic resonance imaging (MRI) and transthoracic exercise echocardiographic scan performed during the same day. Inclusion criteria were: absence of any degree of LV outflow tract obstruction (Doppler gradient on the latest echocardiographic examination ≤20 mm Hg); arm–leg blood pressure gradient ≤20 mm Hg at last examination; no echocardiographic evidence of recoarctation at last outpatient visit (resting pressure gradient at continuous-wave Doppler in the aortic arch ≤25 mm Hg; [7]; absence of a history of coronary artery disease and aortic regurgitation < trivial. Thirty-four patients agreed to take part in the study. Before recruitment in the study, all patients gave written informed consent.

2.1. Exercise echocardiographic evaluation

Before exercise, all patients underwent manual measurement of right arm and right leg blood pressure with a cuff of appropriate size after having rested in the supine position for at least 5 min. A second measure was made after 5 min and results were averaged. Echocardiographic examination was performed with the patient lying in the left supine position, using a Sonos 5500 scan machine (Agilent Technologies, Palo Alto, CA). Continuous-wave Doppler analysis from the suprasternal notch view was used to evaluate systolic pressure gradient (SPG) across the TAA and aortic isthmus using the modified Bernoulli equation ($4v^2$). Pulsed-wave Doppler was used to evaluate flow velocity just proximally to the TAA. The estimate of actual SPG was made using the extended Bernoulli equation ($SPG = 4(V_2^2 - V_1^2)$), subtracting proximal velocity (Fig. 1). Additionally, color Doppler was used to identify the site of flow acceleration. The TAA was considered responsible for the SPG when the increase of flow velocity was detected within 3 cm proximally to the origin of the left carotid artery. A single experienced investigator who was blinded to MRI results made all echocardiographic measurements. After this baseline evaluation, patients were encouraged to exercise in a semi-recumbent position on an electrically braked cycle-ergometer (Ergometrics 900 L, Ergoline, Bitz, Germany). All patients received the same standardized exercise protocol consisting of an initial workload of 20 W/min with subsequent increment of 10 W/min, with a target workload of 100 W. Blood pressure at the right arm was automatically monitored every minute throughout exercise, by temporary replacement of the cuff by a computerized microphone (Ergometrics 900). Within 30 s from the cessation of exercise, we manually measured right arm and right leg blood pressure, and arm–leg pressure gradient was calculated from these values. Systolic pressure gradient through the descending aorta was calculated at baseline and each minute thereafter, throughout the whole exercise phase. Every minute, at least 6 beats were recorded on a standard tape and stored on a magneto-optical disk for further evaluation purposes. The test was considered as completed when heart rate exceeded 160 beats/min, right arm blood pressure was ≥230 mm Hg, or the target workload of 100 W was reached. Maximum test workload was set to 100 W to produce a sub-maximal effort that simulates patient daily life activities, rather than maximal effort. An abnormal blood pressure at rest was defined as a value exceeding the 95th percentile according to sex and age [9], whereas an abnormal response of blood pressure to exercise was defined as a systolic blood pressure ≥200 mm Hg [10,11].

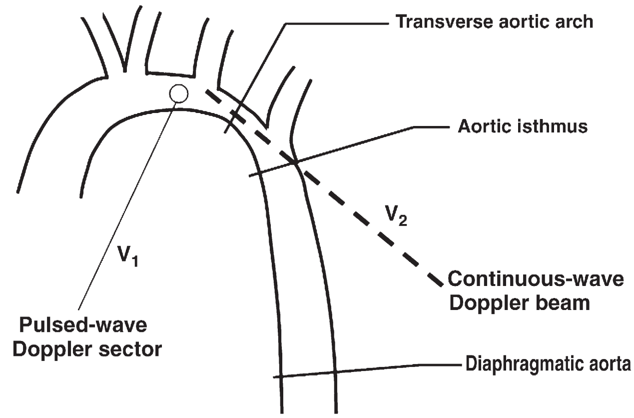

Fig. 1. Scheme of the thoracic aorta, showing both the sites of pulsed-wave and continuous-wave Doppler velocity sampling from the suprasternal view, and the sites of MRI measurement of aortic diameters and flow.

2.2. Magnetic resonance image acquisition

Magnetic resonance imaging was performed with a 1.5-T scanner (Signa; GE Medical Systems, Milwaukee, WI). A phased-array coil (Torsopa; GE Medical Systems) was used. The MRI imaging protocol included an electrocardiogram-gated fast spin-echo double inversion-recovery sequence that was performed in the transverse and sagittal oblique planes. A fast 2-D phase contrast sequence (GE Medical Systems) was used with a *k*-space segmentation of eight lines per segment. During one breath hold, 20 images were obtained, representing the cardiac cycle. Velocity encoded cine MRI images were obtained in an oblique axial plane perpendicular to the aortic lumen at two sites in the descending aorta: 1 cm distal to the AoC repair site and at the level of the diaphragm. A velocity encoding of 400 cm/sec was used to avoid aliasing. Breath-hold cine sequence with steady-state free precession by FIESTA (Fast Imaging Employing Steady State Acquisition; GE Medical Systems, Milwaukee, WI) was performed, covering the whole left ventricle in short axis plane and in four-chamber view. Contrast-enhanced MRI angiography was performed with a three-dimensional dynamic fast spoiled gradient-echo sequence after an