IJC Heart & Vessels 3 (2014) 78–81

Contents lists available at ScienceDirect

IJC Heart & Vessels

journal homepage: http://www.journals.elsevier.com/ijc-heart-and-vessels

Echocardiographic elastic properties of ascending aorta and their relationship with exercise capacity in patients with non-ischemic dilated cardiomyopathy

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ARTICLE INFO

Article history:
Received 1 December 2013
Received in revised form 22 March 2014
Accepted 29 March 2014
Available online 18 April 2014

Keywords:
Heart failure
Aortic distensibility
Aortic stiffness
Dilated cardiomyopathy
Cardiopulmonary exercise test
Ventriculo-arterial coupling

ABSTRACT

Background: : Aortic stiffness, an independent predictor of mortality and cardiovascular events, is common among patients affected by non-ischemic dilated cardiomyopathy (NIDC) and heart failure (HF).
Methods: : A total of 55 patients with diagnosis of NIDC (aged 60 ± 11 years, mean ejection fraction (EF) 35.2% ± 7.7%) admitted consecutively to our department for mild to moderate HF (NYHA class II–III) underwent an echocardiographic study and cardiopulmonary exercise test (CPX). We evaluated elastic properties of ascending aorta, i.e. aortic stiffness and aortic distensibility (mm Hg^{-1}), derived from ascending aorta systolic and diastolic diameter (mm/m^2) measured 3 cm above the valvular plane through 2D-guided M-mode echocardiography.
Results: : Mean aortic stiffness was 15.63 ± 14.53 and aortic distensibility was 2.61 ± 2.39 mm Hg^{-1}. Collected parameters at CPX were peak oxygen consumption (pVO_2) (ml/kg/min), anaerobic threshold (AT) and the slope of the relation between minute ventilation (VE) and carbon dioxide production (VCO_2). Mean pVO_2 was 15.4 ± 3.9 ml/kg/min, VE/VCO_2 ratio at AT was 36.1 ± 6.1. Functional capacity measured through peak VO_2 was found to be directly correlated with aortic distensibility ($r = 0.47$, $p = -0.0002$) and negatively correlated to aortic stiffness index ($r = -0.51$, $p = -0.0001$). These results were the same at multivariate analysis, corrected by age, hypertension, diabetes mellitus and ejection fraction (respectively $r = 0.27$, $p = 0.008$ and $r = -1.75$, $p = 0.0002$).
Conclusions: : HF patients due to NIDC elastic properties of ascending aorta, evaluated by echocardiography, are correlated with a reduced functional capacity.

1. Introduction

The interaction between the heart and the peripheral vasculature is one of the major determinants of cardiovascular performance. The progression of myocardial systolic dysfunction in chronic heart failure (CHF) is characterized by a reduction of left ventricular end-systolic elastance (LVE) and increased arterial elastance (AE), or stiffness [1,2]. The relationship between AE and LVE represents the ventriculo-arterial coupling that is an important determinant of net cardiac performance and cardiac energetics [3]. Ventriculo-arterial coupling is known to progressively increase in CHF due to a reduction in LVE and a raise in AE [4,5]. The failing heart is so very linked to afterload conditions during exercise. Many studies showed a progressive alteration of large-artery function with the increasing severity of CHF [6]. Recent studies have shown that increased aortic stiffness leads to an increased afterload [7] and an impaired ventriculo-arterial coupling [8], thus reducing left ventricular systolic function and exercise capacity in patients with CHF [9,10].

The aim of our study was to demonstrate the possible relationship between elastic properties of ascending aorta, evaluated noninvasively by echocardiography, and clinical functional capacity evaluated by cardiopulmonary test in patients with NIDC.

2. Methods

We evaluated 55 consecutive patients affected by mild to moderate CHF due to NIDC with reduced left ventricular ejection fraction (LVEF < 45%), admitted to Cardiology Department of Spedali Civili of Brescia, Italy. 30 patients (55%) were in NYHA class II and 25 (45%) in NYHA class III. They received standard therapy of CHF in accordance with the European Society of Cardiology guidelines for the treatment of

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http://dx.doi.org/10.1016/j.ijchv.2014.03.009

CHF [11]. NIDC was confirmed by coronary angiography. Every patient had a diagnosis of idiopathic cardiomyopathy.

The authors stated that written informed consent was obtained from each patient and that the study protocol conforms to the ethical guidelines of the 1975 Declaration of Helsinki.

All the patients underwent a complete echocardiography-Doppler examination (VIVID 7 echocardiograph General Electric Medical Systems, Horten, Norway). Left ventricle end-diastolic and end-systolic volumes, left ventricle end-diastolic and end-systolic diameters, ejection fraction and also left ventricular wall thicknesses at end diastole and end systole were measured in accordance with the guidelines [12]. The Doppler method was used to calculate trans-mitral early and late flow velocities (E- and A-wave components) and deceleration time. The ratio of early trans-mitral flow velocity to early mitral annular velocity (E/E′) was measured by tissue Doppler imaging [13]. LV diastolic dysfunction was classified as proposed by the Group on Diastolic Heart Failure [14]. Systolic blood pressure in pulmonary artery (sPAP) was estimated using Doppler echocardiography [15,16].

To evaluate aortic properties of ascending aorta we assessed aortic size at four levels: Valsalva sinuses (VS), sino-tubular junction (STJ), ascending aorta (AA), and aortic arch (AAr) at the end of diastole and systole. So, aortic elastic indexes, i.e. distensibility (AoDis) and stiffness (AoStif) were calculated from the echocardiographically derived thoracic aortic diameters (indexed by body surface area — mm/m^2) and assessed on the basis of a two-dimensional guided M-mode recording of systolic (AoS) and diastolic (AoD) aortic diameters, 3 cm above the aortic valve. AoD was obtained at the peak of the R wave at the simultaneously recorded ECG, and AoS was measured at the maximal anterior motion of the aortic wall; five measurements were averaged for each diameter. The following indexes of aortic elasticity were calculated: AoDis = [2 × (AoS − AoD) / AoD ∗ PP] ($mm\ Hg^{-1}$); (AoStif) = ln(SBP/DBP) / [(AoS − AoD) / AoD] (pure number) where SBP and DBP refer to brachial systolic and diastolic BP respectively, in mm Hg and pulse pressure (PP) was calculated as SBP − DBP [17].

All the patients underwent a cardiopulmonary exercise (CPX) test (Marquette Hellige). It was used as a constant ramp protocol where work rate was increased of 10 W/min. Gas exchange was monitored during the exercise test with a spirometer and a computer metabolic analysis (Medical Graphics). Collected parameters were peak oxygen consumption (pVO_2) expressed in ml/kg/min, anaerobic threshold (AT) expressed in ml/kg/min, the slope of the relation between minute ventilation (VE) and carbon dioxide production (VCO_2). Peak VO_2 was measured as the mean value during the last 30 s of exercise.

All data are given as mean ± standard deviation or number (percentage) of patients. Univariate and multivariate analyses were made to assess a correlation between pVO_2, age, LVEF, hypertension, diabetes and aortic stiffness. Statistical significance was established at a level of $p < 0.05$.

3. Results

Patients were 46 males (83%) and 9 females (17%). Mean age was 60 ± 11 years old. All patients were affected by CHF due to NIDC with mean EF of 35.2% ± 7.7% and were treated with optimized medical CHF-therapy according to current guidelines: ACE-I or ARB (96%), beta blocker (96%), aldosterone receptor antagonist (64%) and diuretics (70%).

All the other characteristics and all the echocardiographic parameters are summarized respectively in Tables 1 and in 2.

Peak VO_2 at CPX was 15.4 ± 3.9 ml/kg/min, VE/VCO_2 ratio at AT was 36.1 ± 6.1; mean respiratory exchange ratio (RER) was 1.1 ± 0.14 thus meaning that a maximum metabolic effort was performed. All the other parameters evaluated during CPX were summarized in Table 3.

Mean AoDis was 2,61 ± 2.39 $mm\ Hg^{-1}$ and mean aortic stiffness 15,63 ± 14,53.

Table 1
Characteristics of the study population of mild- to moderate CHF (n = 55).

Age (years)	60 ± 11
Men/women	46 (83%)/9 (17%)
Body surface area (BSA) (m^2)	1,96 ± 0,2
Systolic blood pressure (mm Hg)	119 ± 15.2
Diastolic blood pressure (mm Hg)	74,9 ± 8,2
Heart rate (bpm)	68,4 ± 10,8
Arterial hypertension	8 (15%)
Chronic obstructive pulmonary disease (COPD)	6 (10%)
Diabetes mellitus	9 (18%)
Smokers	3 (5%)
Dyslipidemia	17 (30%)

Data are presented as the mean value ± SD, or number (percentage) of patients.

At univariate analysis only pVO_2 was found to be directly correlated with AoDis (r = 0.47, CI 0.25–0.66, p = 0.0002) and negatively correlated to AoStiff (r = −0.51, CI −0.68 to −0.20, p = 0.0001) [Figs. 1 and 2]. The correlations were also demonstrated at multivariate analysis, corrected by hypertension, diabetes, age and ejection fraction (respectively r = 0.27, p = 0.008 and r = −1.75, p = 0.0002). In Table 4 was shown the predictors of peak volume oxygen consumption.

4. Discussion

This study demonstrates that aortic stiffness (measured non-invasively using 2D guided M-mode echocardiography) is an independent predictor of exercise capacity in patients with CHF and NIDC.

Our results are consistent with other reports that showed a correlation between elastic properties of ascending aorta and functional capacity in CHF patients using different echocardiographic methods [9,10]. The echocardiographic method used to evaluate aortic stiffness in CHF patients is simple and easy to acquire and is not dependent on pulse wave Doppler echocardiography.

An impaired aortic compliance is frequent among CHF patients [18–20] and we showed that aortic stiffness is inversely related to peak VO_2, which is a major predictor of mortality among CHF patients [21]. Our study cannot establish a causal relation between aortic stiffness and decreased functional capacity. Actually, the elastic proximal aorta is the main region that is affected by aging, hypertension, diabetes or LV diastolic dysfunction. Therefore an increased aortic stiffness may represent a marker of more advanced disease in CHF patients. Despite that, several pathophysiological mechanisms suggest a possible causal

Table 2
Main echocardiographic parameters of the study population (n = 55).

End diastolic diameter (EDD) (cm)	6.66 ± 0.79
End systolic diameter (ESD) (cm)	5.32 ± 0.95
End diastolic volume (EDV) (ml)	183.27 ± 82.58
End systolic volume (ESV) (ml)	132.73 ± 94.38
End diastolic volume indexed (EDVi) (ml/m^2)	74.36 ± 49.23
Left ventricular ejection fraction (LVEF) (%)	35.2 ± 7.7
Posterior wall thickness (PWT) (cm)	0.95 ± 0.14
Interventricular septum thickness (IVT) (cm)	1.05 ± 0.18
Left ventricular mass (LV mass) (g)	
Valsalva sinuses (VS) (mm)	37.23 ± 6.37
Sino-tubular junction (STJ) (mm)	35.32 ± 2.56
Ascending aorta (AA) (mm)	42.93 ± 4.65
Aortic arch (Aar) (mm)	33.28 ± 2.96
Aorta in systole (AoS) (mm)	33.24 ± 5.03
Aorta in diastole (AoD) (mm)	34.95 ± 5.1
Aortic distensibility ($mm\ Hg^{-1}$)	2.61 ± 2.39
Aortic stiffness index	15.63 ± 14.53
E/A ratio	0.75 ± 0.20
Deceleration time (DT) (msec)	244.6 ± 80.4
E/e′ ratio	14.7 ± 6.26
Pulmonary artery systolic pressure (mm Hg)	30.5 ± 5.5
Left atrium diameter (cm)	4.63 ± 0.9
Left atrium area (cm^2)	25.01 ± 7.57

Data are presented as the mean value ± SD, or number (percentage) of patients.