IJC Heart & Vessels 4 (2014) 102–107

Contents lists available at ScienceDirect

IJC Heart & Vessels

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

Atrial fibrillation in severe aortic valve stenosis — Association with left ventricular left atrial remodeling ☆

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ARTICLE INFO

Article history:
Received 3 June 2014
Accepted 30 June 2014
Available online 6 July 2014

Keywords:
Aortic stenosis
Atrial fibrillation
Diastolic function
Remodeling
Surgery

ABSTRACT

Background: Atrial fibrillation (AF) is common in patients with aortic stenosis (AS) although the exact mechanism is unclear. The purpose of this study was to investigate echocardiographic characteristics among patients with severe AS and AF and to identify factors associated with the development of new-onset AF after aortic valve replacement (AVR).
Methods: 125 patients with severe AS and ejection fraction >40% scheduled for AVR were evaluated preoperatively and 3, 6, 9 and 12 months postoperatively with electrocardiography (ECG) and echocardiography, and Holter-ECG analysis was performed after 3 and 12 months. The primary endpoint was new-onset AF defined as an episode of AF exceeding 30 s, on the ECG or Holter-ECG and/or patients hospitalized due to AF.
Results: AF was present in 19 patients prior to AVR, compared to patients in sinus rhythm AF patients had increased NT-proBNP, increased left atrial (LA) volume (61 ± 21 vs. 47 ± 17 ml/m^2, $p = 0.002$), reduced global longitudinal left ventricular strain (-13.1 ± 3.7 vs. -16.0 ± 3.5, $p = 0.002$) and presented more often with a restrictive filling pattern (37% vs. 10%, $p = 0.002$). During follow-up 23 patients developed new-onset AF; predictors were LA volume, restrictive filling pattern, NT-proBNP, E/e′ and systolic blood pressure. After correcting for age and LA volume index, a restrictive filling pattern and systolic blood pressure remained associated with new-onset AF.
Conclusions: The presence of preoperative AF and development of new-onset AF after AVR is associated with restrictive filling pattern and LA dilatation in patients with severe AS.
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1. Introduction

Atrial fibrillation (AF) is the most common sustained cardiac arrhythmia affecting up to 1.5–2% of the general population [1] and more than 9% in patients with aortic stenosis (AS) [2] and is independently associated with increased mortality and serious cardiovascular events such as ischemic stroke, systemic thromboembolism, and heart failure [3].

AS is a progressive disease involving lipid accumulation, extracellular matrix deposition and inflammatory response of the valve apparatus leading to chronic left ventricular (LV) and left atrial (LA) pressure overload. Due to direct effects on the myocardium, pressure overload leads to LV hypertrophy and myocardial fibrosis, which increase myocardial stiffness. The consequences of these pathophysiological processes are diastolic dysfunction, increased LV filling pressures, increased LA pressure and LA dilatation.

Systemic hypertension (LV pressure overload) especially when associated with LV hypertrophy are considered predictors of AF [4–6]. The exact pathophysiological mechanisms, however, underlying the relation between elevated afterload and the development of AF remain unclear. The association of AS with AF is thus not fully understood. The aim of this study was to investigate echocardiographic and biomarker characteristics among patients with severe symptomatic AS and AF and to evaluate factors able to predict the development of new-onset AF after aortic valve replacement (AVR).

2. Methods

The present investigation is a sub-study of a prospective single center, randomized study to evaluate the effect of candesartan on top of conventional treatment on reverse remodeling in consecutive patients undergoing AVR for symptomatic AS. The study was registered with the National Board of Health and the Danish Data Protection Agency

☆ The trial has been registered at www.clinicaltrial.gov with Identifier: NCT00294775.
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http://dx.doi.org/10.1016/j.ijchv.2014.06.006
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and approved by the local ethics committee. All patients gave written informed consent. ClinicalTrials.gov Identifier: NCT00294775. The study design and the results regarding the effect of candesartan on regression of LV hypertrophy have previously been published [7]. In brief, we enrolled patients aged >18 years with symptomatic severe AS (estimated aortic valve area <1 cm^2) scheduled for AVR at Odense University Hospital, Denmark between February 2006 and April 2008. Patients with LV ejection fraction <40%, s-creatinine >220 μmol/l, previous aortic valve surgery, planned additional valve repair/replacement, infective endocarditis, predominant aortic valve regurgitation, or ongoing treatment with an angiotensin converting enzyme inhibitor or an angiotensin receptor blocker were excluded.

2.1. Echochardiography

All echocardiograms were performed by the same experienced operator on a GE Vivid 5 ultrasound system (GE Medical System, Horten, Norway), the day prior to surgery. Echocardiograms were digitally stored and later analyzed completely blinded for all clinical and survival data. Aortic valve area was estimated by quantitative Doppler ultrasound using the continuity equation. Peak flow velocity across the valve was determined in the window where the highest velocity could be recorded using continuous wave Doppler with the cursor as parallel as possible with the flow across the valve. Peak transvalvular gradient was estimated using the modified Bernoulli equation. Finally, the peak systolic flow velocity in the outflow tract was estimated with pulsed wave Doppler [8].

As a measure of LV hypertrophy, LV mass was estimated according to the joint recommendations of the American (ASE) and European (EAE) associations of echocardiography using Devereux's formula [9]. Relative wall thickness was calculated using the formula 2 × posterior wall thickness / LV internal diameter in diastole [10]. LV ejection fraction was estimated using Simpson's biplane method. Longitudinal LV systolic function was assessed using peak systolic mitral annular motion assessed with tissue Doppler imaging with the Doppler sample volume placed in the septal mitral valve annulus. Global longitudinal strain (GLS) was analyzed using EchoPAC PC 08 (GE Medical system, Horten, Norway) speckle tracking software 2-D. GLS was determined as the magnitude of strain at the aortic valve closure, and systolic strain rate (SR_S) was determined as the maximal negative SR value during the ejection phase. Both parameters were assessed in all 3 apical planes, and the mean values (GLS_{mean}, SR_{smean}) were calculated. Frame rate was kept as high as possible with a minimum frame rate of 70/s.

Mitral inflow was assessed in the apical four-chamber view using pulsed-wave Doppler with the sample volume placed at the tips of mitral leaflets during diastole. From the mitral inflow profile, the E- and A-wave peak velocities and deceleration time were measured. Doppler tissue imaging of the mitral annulus, was used in the septal annulus to measure the early diastolic e′–velocity. The E/e′–ratio was used as a non-invasive marker of LV filling pressures [11]. E/e′ greater than 15 was considered consistent with increased filling pressure. Diastolic filling pattern and restrictive filling pattern was categorized according to European Association of Echocardiography (EAE) guidelines [12].

LA volume was assessed using the area length method [9] from the apical four and two-chamber views. Measurements were obtained in end-systole from the frame preceding mitral valve opening, and the volume was indexed for body surface area. Patients were considered to have severe LA dilatation if left atrial volume index (LAVi) was 40 ml/m^2 or greater.

2.2. Assessment of atrial fibrillation

Patients were examined at 3, 6, 9 and 12 months after AVR and underwent a 12-lead ECG for 2 min in the supine position. In addition, 24-hour Holter monitoring was performed at 3 months and 48-hour Holter monitoring at 12 months follow-up using a Reynolds Medical Tracker 3 recorder and the Pathfinder 700 analyzer (Reynolds Medical Limited, U.K.). All ECGs were interpreted by the same experienced cardiologist blinded for all clinical, echocardiographic and survival data. An episode of irregular heart rhythm without definite p-waves and with a minimum duration of 30 s was considered as AF according to guidelines [13]. Short episodes of supraventricular tachycardia (SVT) <30 s were recorded separately. AF episodes occurring within 30 days of valve replacement were regarded as postoperative AF and not included as an endpoint.

Table 1
Baseline characteristics of the study population.

	Atrial fibrillation prior to AVR n = 19	No atrial fibrillation prior to AVR n = 106	
Age (y)	76 ± 8	72 ± 9	0.10
Sex (male)	13 (68)	66 (62)	0.61
NYHA I/II/III/IV	6/8/5/0	17/57/31/1	0.43
Diabetes mellitus	3 (16)	16 (15)	0.94
Ischemic heart disease	1 (5)	22 (21)	0.11
EuroScore	6.5 ± 2.0	5.6 ± 2.0	0.08
Aortic valve area (cm^2)	0.76 ± 0.29	0.82 ± 0.27	0.41
Aortic valve mean gradient (mmHg)	33 ± 17	37 ± 17	0.42
Valvulo-arterial impedance (ml/mmHg)	5.3 ± 1.7	4.8 ± 1.5	0.28
Ejection fraction (%)	54 ± 7	54 ± 8	0.75
Left ventricular end-diastolic volume (ml)	108 ± 39	110 ± 33	0.82
Left ventricular mass index (g/m^2)	144 ± 46	128 ± 39	0.13
Relative wall thickness	0.63 ± 0.09	0.59 ± 0.15	0.31
Left atrial volume index (ml/m^2)	61 ± 21	47 ± 17	0.002
E-velocity (m/s)	0.96 ± 0.26	0.77 ± 0.21	0.001
Deceleration time (ms)	176 ± 67	202 ± 57	0.03
Deceleration time <140 ms	7 (37)	11 (10)	0.002
e′ (cm/s)	7.2 ± 2.4	5.4 ± 0.1.3	<0.0001
s′ (cm/s)	5.1 ± 1.5	5.8 ± 1.5	0.04
E/e′	14.4 ± 5.8	15.0 ± 5.2	0.69
Global longitudinal strain (%)	−13.1 ± 3.7	−16.0 ± 3.5	0.002
Systolic strain rate (s^{-1})	−0.75 ± 0.27	−0.85 ± 0.18	0.049
Log-NTproBNP	7.2 ± 1.0	6.1 ± 1.3	0.0002
Fibulin-1 (μg/ml)	104 ± 34	86 ± 32	0.02

Abbreviations: NYHA New York Heart Association.

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