International Journal of Cardiology 165 (2013) 512–518

Contents lists available at SciVerse ScienceDirect

International Journal of Cardiology

journal homepage: www.elsevier.com/locate/ijcard

Left ventricular torsion abnormalities in patients with obstructive sleep apnea syndrome: An early sign of subclinical dysfunction

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ARTICLE INFO

Article history:
Received 25 April 2011
Received in revised form 2 July 2011
Accepted 6 September 2011
Available online 2 October 2011

Keywords:
Obstructive sleep apnea syndrome
Echocardiography
Left ventricular function
Speckle tracking imaging
Aortic wall strain
Aortic wall stiffness

ABSTRACT

Background: Previous echocardiographic studies using tissue Doppler imaging (TDI) and speckle tracking imaging (STI) have demonstrated that obstructive sleep apnea syndrome (OSAS) patients may develop subclinical left ventricular (LV) systolic and diastolic dysfunction. Our purpose was to evaluate the impact of OSAS on LV torsion dynamics and aortic stiffness by using TDI and STI echocardiography.
Methods: Forty-two patients with OSAS and no comorbidities were studied. They were classified into mild and severe OSAS according to the apnea-hypopnea index (AHI). Thirty-five healthy subjects were selected as controls. Fifteen patients with severe OSAS underwent chronic nocturnal nasal continuous positive airway pressure (CPAP) therapy. Standard echocardiographic parameters were assessed. Global LV longitudinal strain (LS), radial and circumferential strain were determined by STI. Averaged LV rotation and rotational velocities from the base and apex were obtained and used for calculation of LV torsion (LVtor). Mitral annular velocities and aortic wall velocities and strain (AoS) were also obtained by TDI.
Results: Severe OSAS had decreased LS compared with control subjects. LVtor increased significantly in severe OSAS compared to normals ($p<.001$) as a result of a predominant increase in apical rotation and was independently related to AHI and AoS in a multiple stepwise linear regression model. The group treated with CPAP had a significant decrease in LVtor and aortic stiffness index and significant increase in LS and AoS.
Conclusions: LVtor, LS and AoS were identified as parameters demonstrating an association between LV dysfunction, aortic stiffness and severity of OSAS independently of other possible factors or comorbidities.

1. Introduction

Obstructive sleep apnea syndrome (OSAS) is a sleep-related breathing disorder involving an increased risk of cardiovascular disease. OSAS has been recognized to impose several adverse effects on the cardiovascular system, to be associated with heart failure, and to represent an independent risk factor for hypertension [1,2]. Left ventricular (LV) hypertrophy and arterial dysfunction in OSAS have been reported even in the absence of systemic hypertension [3–7]. Previous echocardiographic studies using tissue-Doppler imaging (TDI) and speckle tracking imaging (STI) have demonstrated that OSAS patients may develop subclinical LV systolic and diastolic dysfunction [8–10]. Recently, STI has been validated as a noninvasive technique to quantify LV torsion dynamics, including the assessment of systolic torsion and diastolic filling versus magnetic resonance tagging [11–13]. Our purpose was to evaluate the impact of OSAS on LV torsion parameters and the relationship between these functional changes and aortic stiffness.

2. Methods

2.1. Population

A cohort of forty-two sleep apnea patients without systemic hypertension was identified among seventy-six people screened in the sleep disorder laboratory and examined with polysomnography and echocardiography. Thirty-five healthy subjects were matched for age, gender, and body mass index, and selected as controls. Subjects completed questionnaires on sleep habits and general health, and had height, weight, and blood pressure measured. A questionnaire to assess the Epworth sleepiness scale [4], which is a rapid, validated method for screening daytime sleepiness, was completed by all subjects. The inclusion criteria for control subjects were: [1] apnea-hypopnea index (AHI) <5 and [2] Epworth sleepiness scale <10. OSAS patients had to fulfill the following inclusion criteria: [1] AHI ≥15, [2] Epworth sleepiness scale ≥10, and [3] no previous treatment for OSAS. Patients with any of the following were excluded: central sleep apnea, systemic hypertension, any known cardiac disease (by history, physical examination, electrocardiogram, chest radiography, conventional stress testing, and echocardiography), atrial fibrillation, obstructive or restrictive lung disease (demonstrated on pulmonary function testing), pulmonary hypertension due to identifiable causes (except OSAS); chronic renal and hepatic diseases, diabetes mellitus (on history, or two random blood glucose levels >125 mg/dL), connective-tissue disease, and morbid

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doi:10.1016/j.ijcard.2011.09.030

obesity (body mass index >40 kg/m^2). Patients with poor echocardiographic window were also excluded when <80% of the endocardial border was adequately visualized.

2.2. Standard echocardiography

All patients underwent transthoracic echocardiography (Vivid E9 ultrasound system, GE, Horten, Norway). Measurements of cardiac chambers were made by transthoracic echocardiography according to established criteria [14]. LV ejection fraction by modified Simpson method and mass index were estimated [14,15]. Peak early (E) and late (A) diastolic velocities, deceleration time, left ventricular isovolumic relaxation time, myocardial performance index, and right ventricular systolic pressure were obtained using standard Doppler practices [16-18].

2.3. LV tissue Doppler imaging

The general principles that underlie the TDI modalities have been described previously [19]. Mitral annulus velocities (S_a, E_a, A_a) were measured on the transthoracic four chamber views. E/E_a ratio was used to estimate LV filling pressures and diastolic function [19–21].

2.4. LV speckle tracking imaging

Two-dimensional strain was measured as previously described [22,23]. After tracing endocardial border at an end-systolic frame, the operator could validate the tracking quality and adjust the endocardial border or modify the width of the region of interest. Aortic valve opening and closure were selected on pulsed-wave Doppler tracings recorded from the LV outflow tract. Frame rate ranged from 60 to 100 frame/s, and three cardiac cycles were stored in cineloop format for offline analysis. Longitudinal LV strain was defined as the average of negative longitudinal strains of 6 segments of the septal and lateral walls in the apical 4-chamber view (Fig. 1). Average radial and circumferential strain of 6 mid-LV segments was determined in the mid-short-axis view. The assessment of LV rotation by 2D speckle-tracking strain imaging required the acquisition of the LV short-axis at the basal and apical level. The basal level was defined as that which showed the mitral valve tip and the apical level as that which was just proximal to the level with LV cavity obliteration at end-systole. The time rotation curves were displayed along the cardiac cycle. Counterclockwise rotation was conventionally marked as positive value and clockwise rotation as negative value when viewed from the LV apex. Peak apical and basal rotation and peak LV torsion were obtained. LV torsion or twist (LVtor) was defined as the net difference (in degrees) of apical and basal rotation at isochronal time points. Normalized torsion was torsion divided by LV ventricular diastolic longitudinal length between apex and mitral plane. Peak diastolic untwisting velocity and time to peak untwisting velocity were measured. Twisting rate (TR) was defined [13] as (peak LVtor − LVtorEarlySystole)/(time difference between these two events). Untwisting rate (UTR) was defined as (peak LVtor − LVtorMVO)/(time difference between these two events) where MVO is mitral valve opening. The analysis of strain and rotation parameters was performed offline using customized computer software (EchoPAC, version 9.0, GE Ultrasound).

2.5. Aortic function

The elastic properties of the aorta were indexed by calculation [24–26] of aortic distensibility (D), stiffness index (SI), and pressure-strain elastic modulus (E_p) as $D = 2(A_s - A_d)/[A_d\ (P_s - P_d)]$, $SI = \ln(P_s/P_d)/(A_s - A_d)/A_d$, and $E_p = (P_s - P_d)/[(A_s - A_d)/A_d]$, respectively, where A_s = aortic diameter at end-systole, A_d = aortic diameter at end-diastole, P_s = systolic blood pressure, P_d = diastolic blood pressure, and ln = natural logarithm.

Aortic wall TDI velocities have been obtained as previously described [27]. By marking a region of interest on the 2D image in the anterior aspect of the ascending aorta at the same point as in M-mode measurements, velocities throughout the cardiac cycle for this area can be determined. Offline analysis of the velocity data sets was performed using dedicated software (EchoPAC, version 9.0, GE Ultrasound). TDI tracing displayed accelerated expansion of the aortic wall followed by a slow deceleration, a plateau and then a rapid deceleration into diastole. This trace represents the mean of the instantaneous velocity spectrum. Systolic maximum wall expansion velocity (AoSvel, cm/s), wall contraction early diastolic velocity (AoEvel, cm/s), and wall peak systolic radial strain (AoS,%) were derived.

2.6. Sleep study

All patients underwent overnight polysomnography using a standard technique. A device measuring thoracic and abdominal respiratory movements and combined oronasal flow (PM unit, MAP, Martinsried, Germany) was attached to the patient. The following seven channels were monitored: [1] a flow sensor for joint registration of nasal and oral breath flow; [2] a laryngeal microphone; [3] a three-channel ECG; [4] one stress-sensitive belt for the thorax; [5] one stress-sensitive belt for the abdomen; [6] a positional sensor for determination of body position; [7] a pulse oximeter. A customer software (Poly-Mesam, version 1.42, MAP, Martinsried, Germany) gave the possibility to manually and/or automatically score the events on the raw data and analyze the graphical display.

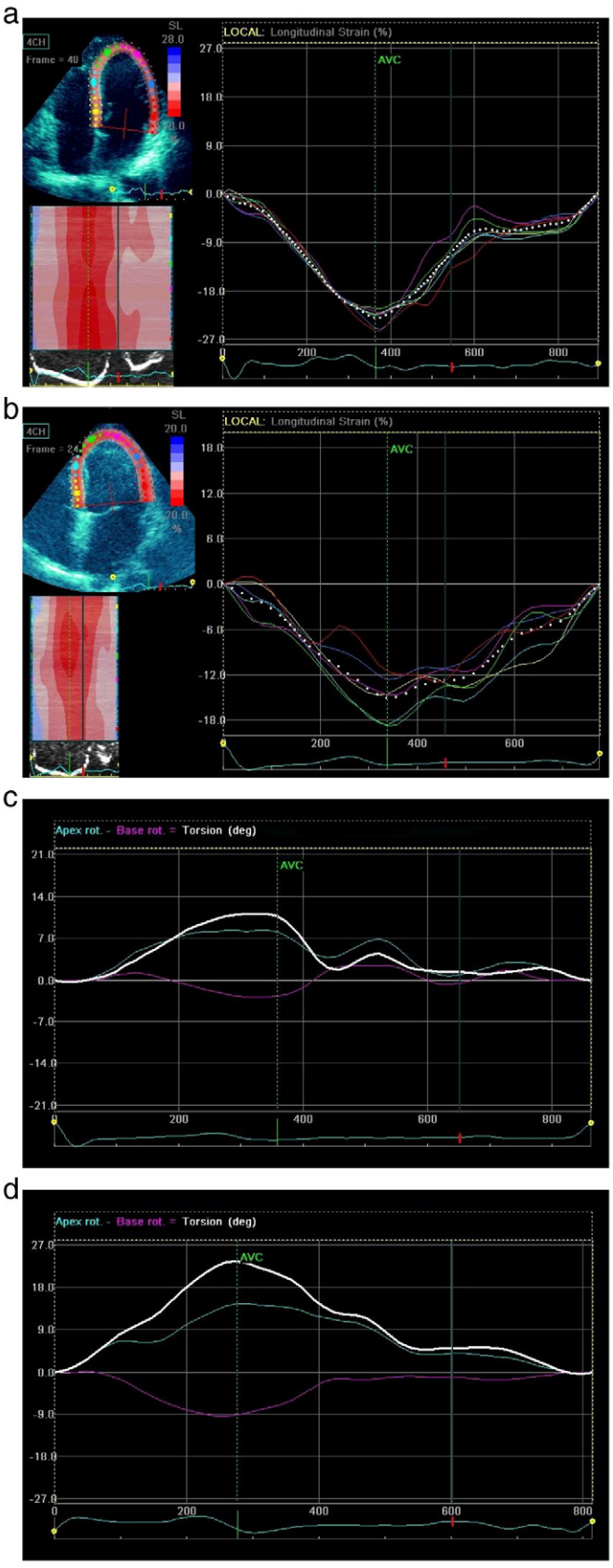

Fig. 1. Abnormal LV strain and torsion in OSAS patient compared to a normal control. 1a. Global LS (24%, white dotted line) in a normal control. 1b. Reduced global LS (13%, white dotted line) in a OSAS patient. 1c. Torsion (12, white solid line) in a normal control. 1d. Increased torsion (26, white solid line) in a OSAS patient. LS = longitudinal strain.