Journal of Clinical & Experimental Cardiology

Maria et al. J Clinic Experiment Cardiol 2011, 2:10
DOI: 10.4172/2155-9880.1000162

Research Article Open Access

The Prevalence of Left Ventricular Hypertrophy and Altered Geometry in Patients with Right Ventricular Diastolic Dysfunction

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Abstract

Background: Left ventricular diastolic dysfunction (LVDD) associated with left ventricular hypertrophy (LVH) has been reported to play a major role in cardiovascular heart failure. Although, right ventricular diastolic dysfunction (RVDD) in patients with LVH was known to be clinically relevant, no systematic study had been performed regarding this relationship between left ventricular (LV) geometry associated with LVH and RVDD. The goal of this study was to evaluate an association between RVDD; LVDD, LVH and LV altered geometry for early diagnosis of heart failure.

Methods and Results: Out of 426 patients, 396 patients (93%) were found to have both RVDD and LVDD. In this cohort, LV concentric geometry (LVCG) was identified in 138 patients (32.3%). RVDD and LVDD were diagnosed by measurement of E/A and tissue doppler imaging (E'/A') for mitral and tricuspid valves. The mean value of mitral E/A in the LVCG group was lower than that of controls; 0.63±0.03 vs. 1.44±0.03, p<0.02. Mitral E'/A' was also lower in LVCG than that of controls; 0.60±0.02 vs. 1.36±0.03, p<0.01. Similarly, E/A of Tricuspid valve in patients with LVCG was found to be lower than E/A of Tricuspid valve of controls; 0.52±0.07 vs. 0.69±0.02, p<0.001 and E'/A' of tricuspid valve was lower than that of controls; 0.44±0.02 vs. 0.63±0.06, p<0.05.

Conclusions: This study demonstrated that simple doppler trans-valvular inflow parameters and tissue doppler imaging may identify patients with biventricular diastolic dysfunction. This finding was more often observed in patients with LV concentric geometry, a pattern associated with an increased risk of cardiovascular events.

Keywords: Right ventricular diastolic dysfunction; Left ventricular diastolic dysfunction; Left ventricular concentric geometry; Left ventricular hypertrophy

Background

Chronic LV pressure or volume overload hypertrophy (LVH) leads to LV remodeling [1,2] the first step toward heart failure, causing impairment of both diastolic and systolic function [3,4]. As demonstrated in many epidemiological studies, LVH is a well-known and independent predictor of morbidity and mortality for heart failure (HF) [5,6]. It is one of the powerful risk factors for ventricular arrhythmias, sudden death and congestive heart failure (CHF) [7-10]. Therefore, left ventricular diastolic dysfunction (LVDD) is a frequently considered parameter for LV function evaluation. Although, right ventricular diastolic dysfunction (RVDD) has been reported in these patients, cardiologists often pay little attention to RVDD. It is well known that RVDD is associated right ventricular volume and pressure overload. We often observe patients with right sided CHF in association with LVDD in clinical practice. Therefore, we assessed RVDD in patients with LVDD and altered LV geometry. The common denominator for LV and RV function is the septum which constitutes 40% of LV mass. Furthermore, both the ventricles are bound together by spiral muscle bundles of the septum that encircle them in a complex interlacing fashion making LV and RV highly interdependent. Due to this interdependence, RV dysfunction associated with altered LV geometry and LVDD may play a role in heart failure morbidity and mortality. Furthermore, RVDD associated with LVDD may predict adverse prognosis [6,11]. This study was designed to assess relationship between LV geometry, mass and RVDD and their interdependence mediated by the unique structure of the septum.

Patients and Methods

A population of 7000 consecutive patients underwent comprehensive echocardiography and electrocardiography in our heart station in Brindisi over a period of four years. All patients underwent medical record review. All echocardiograms were performed and interpreted by a cardiologist using an unic echocardiographic instrument (GE Vivid 3 Expert) according to a standardized protocol and were reviewed by another cardiologist. An assessment for valvular disease was performed by two-dimensional echo, color & doppler imaging.

In this group of 7000 patients, 426 patients were found to have RVDD diagnosed by examination of tricuspid inflow profile and tricuspid annulus imaging at the lateral wall of RV according to American Society of Echocardiography recommendations [12,13]. Patients with LV ejection fraction < 50%, wall motion abnormalities, severe valvular disease atrial fibrillation, right ventricular enlargement (RVE) or right atrial enlargement (RAE) and pulmonary hypertension

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Received September 29, 2011; **Accepted** November 17, 2011; **Published** November 21, 2011

Citation: Maria M, Sharma RK, Ciccone MM, Reddy HK, Palmiero (2011) The Prevalence of Left Ventricular Hypertrophy and Altered Geometry in Patients with Right Ventricular Diastolic Dysfunction. J Clinic Experiment Cardiol 2:162. doi:10.4172/2155-9880.1000162

were excluded. Pulmonary hypertension was estimated from the tricuspid regurgitation velocity by modified Bernoulli equation. Clinical characteristics of patients are listed on (Table 1).

Pulsed-wave doppler examination of mitral inflow as well as tissue doppler imaging of the mitral annulus at lateral wall of LV was performed in each subject to determine LVDD as previously described and validated [14-16]. These variables were used to categorize LV diastolic function as normal, impaired relaxation with normal or near-normal filling pressures (grade I); impaired relaxation with moderate elevation of filling pressures, "pseudonormal filling" (grade II); and impaired relaxation with marked elevation of filling pressures, "restrictive filling"(grade III) [14,15].

The mitral inflow early filling velocities (E) to atrial filling velocity (A) ratio were used for initial categorization of LV diastolic dysfunction. We considered 3 different criteria: E/A ratio <0.75, deceleration time (DT) > 240 ms and isovolumic relaxation time >100 m.sec and when 2 out of 3 criteria were present, we assessed a grade-I LVDD. The diagnosis was confirmed by tissue doppler Imaging (TDI); E'/A' ratio < 0.75. If the E/A ratio was in the normal range (0.75 to 1.5) and the deceleration time was >140 ms, we used tissue doppler indexes to determine if filling was normal (normal diastolic function) or "pseudonormal" and if E'/A' was <0.75 called as grade II LVDD. We didn't perform E/A with valsalva or pulmonary venous flow to record simultaneous measurements. If the E/A ratio was >1.5 and the deceleration time was <140 ms, and Tissue Doppler E'/A' was >1.5 we considered diagnosis "restrictive filling" LVDD.

Patients studied : 7.000
Patiens enrolled with RV impaired filling : 426
Left ventricular ejection fraction > 50%: 426 (100%)
Males: 189 (44%)
Females: 237 (55,6%)
Mean age : 66±13 years
Height: 167±12 cm.
Weight 81±18 kg.
Body surface area (BSA): 1.79±0,7 m²
LV Diastolic dysfunction(DD): 396 (93%)
Left ventricular hypertrophy(LVH): 180 (42,2%)
LV concentric geometry : 138 (32,4%)
LV eccentric geometry : 108 (25,3%)
Myocardial infarction: 21 (4,9%)
Hypertensive patients: 287 (67,3%)
Diabetic patients : 123 (28,8%)
Hypercholesterolemic patients : 219 (51,4%)
Smokers : 28 (6,5%)
Former smokers : 110 (25,8%)

Table 1: Clinical characteristics of patients.

E/A and E'/A' Mitral and Tricuspid	*RVDD +LVDD(138) **LVCG Group	RVDD & No LVDD (30) Control Group
Mitral E/A Mitral E'/A'	0.63±0.03 0.60±0.02	1.44±0.03 p<0.02 1.36±0.03 p<0.01
Tricuspid E/A Tricuspid E'/A'	0.52±0.07 0.44±0.02	0.69±0.02 p<0.001 0.63±0.06 p<0.05

E Early filling velocity, A-Atrial Filling velocity
E' Early filling by Tissue Doppler Imaging, A'-Atrial filling by tissue Doppler Imaging
E/A <0.75 suggestive of Diastolic dysfunction
*Higher prevalence of Biventricular diastolic dysfunction in LVCG
** LVCG: Left Ventricular concentric Geometry

Table 2: Trans valvular Flow Velocities and Tissue Doppler Imaging data.

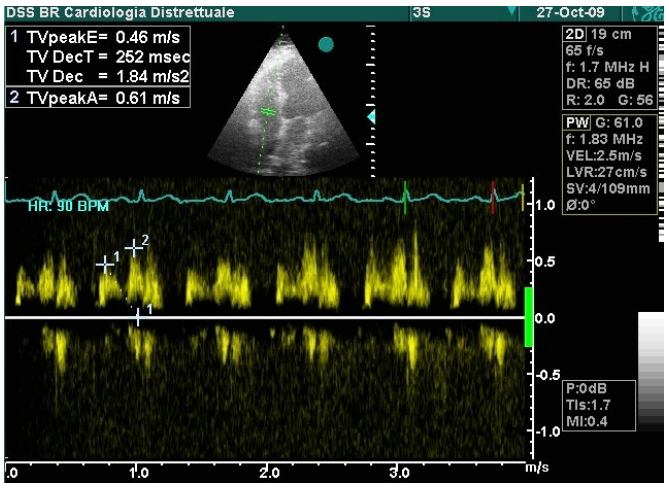

Figure 1: Examination of tricuspid Doppler inflow profile at the lateral wall of RV according to American Society of Echocardiography recommendations.

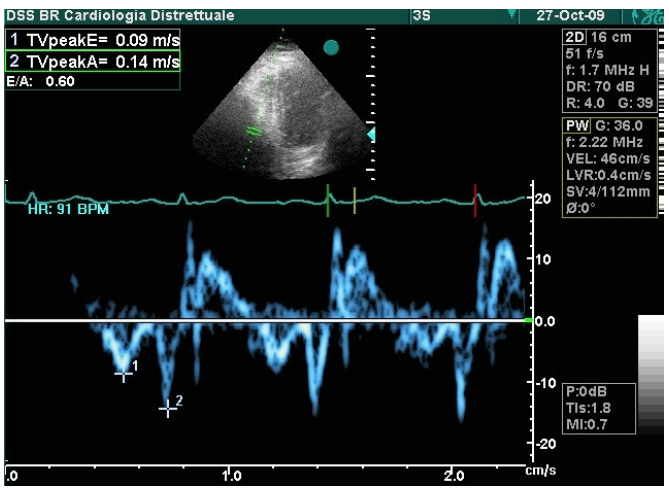

Figure 2: Examination of tricuspid Doppler annulus tissue imaging at the lateral wall of RV according to American Society of Echocardiography recommendations.

The tricuspid inflow early filling velocity (E) to atrial filling velocity (A) ratio was used for initial categorization of RV diastolic dysfunction. If the E/A was <0.75, deceleration time (DT) > 240 ms or isovolumic relaxation time >100 m.sec was present, we called RV diastolic dysfunction as illustrated in (Figure 1) and the diagnosis in all patients was confirmed by TDI doppler E'/A' ratio < 0.75 as shown on (Figure 2). LVDD or RVDD was diagnosed as indeterminate if sufficient doppler parameters were not obtained for technical reasons or if E–A fusion was present. All patients had a complete estimation of LVEF, by M-mode, according to modified Quinones formula, quantitative two-dimensional (biplane Simpson method) and semi-quantitative two-dimensional, visual estimation [14]. We considered that LVEF, by these methods, was highly correlative with magnetic resonance.

LVH and LV geometry were determined by LV mass index and relative wall thickness of LV respectively. LV hypertrophy (LVH) is an increased LV mass indexed by body surface area with 134 g/sq meter in men and 110 g/sq meter for women [17]. LV mass index was also determined to classify LVH by Devereaux formula [18]. We used LV

relative wall thickness (RWT) to differentiate concentric vs. eccentric geometry of left ventricle. LV relative wall thickness was calculated by formula as: left ventricle RWT=IVS+PW/LVEDD**.** If left ventricular RWT was greater than 0.45; it was classified as concentric geometry of LV and if less than 0.34; classified as eccentric geometry [19].

Based on this LV geometry and mass, 3 different patterns of LV geometry were defined as concentric remodeling (concentric geometry with normal mass); concentric hypertrophy (concentric geometry with increased mass) and **e**ccentric hypertrophy (eccentric geometry with increased mass). All subjects underwent history and physical examination to exclude co-morbid conditions. Age, gender, clinical status and BP were obtained in all participants. A detailed physical examination was conducted to exclude cardiac co-morbidities. Measurements also included height, weight and BMI. All study subjects underwent a routine chemistry panel, including fasting glucose, electrolytes and creatinine. All subjects had two-dimensional echocardiogram with good image quality.

Statistical analysis

Continuous variables were summarized as mean values ± SD and categorical data as a percent of the group total. The relationships between two different groups of patients for all the above-mentioned echo findings were assessed by the Chi-square test for non-parametric analysis and Student's t-test for parametric analysis and multivariate analysis was performed too. A p value < 0.05 was considered significant.

Results

Out of 7000 consecutive patients, 426 presented RVDD. 396 patients (93%) presented both RVDD and LVDD, the other 30 (7%) having only RVDD without LVDD, were used as control group. As described in methodology, LV concentric geometry (LVCG) was identified in 138 patients (32.4%). Out of 396 patients, LV eccentric geometry was determined in 108 patients (25.3%) p. <0.02. In this cohort of 396 patients with RVDD and LVDD, 180 patients were found to have LVH by increased LV mass index whereas 150 patients had normal geometry with normal mass (NGNM) as shown in (Figure 3).

RV diastolic dysfunction and LV diastolic dysfunction were assessed by above mentioned criteria, the values of E/A and E'/A' at both mitral valve (MV) and tricuspid valve (TV) inflow are summarized in the (Table 2).

The mean value of mitral E/A in the LVCG group was lower than that of controls; 0.63±0.03 vs. 1.44±0.03, p<0.02. Mitral E'/A' (tissue doppler imaging) was also lower in LVCG than that of controls (mitral E'/A'), 0.60±0.02 vs. 1.36±0.03, p<0.01. E/A of tricuspid valve in patients with LVCG was found to be lower than E/A of tricuspid valve of controls; 0.52±0.07 vs. 0.69±0.02, p<0.001. E'/A' of tricuspid valve was also lower than that of controls; 0.44±0.02 vs. 0.63±0.06, p<0.05. The mean value of mitral E/A was lower in LVCG than NGNM group; 0.63±0.03 vs. 0.75±0.02, p<0.01. Mitral E'/A' (tissue doppler imaging) was lower in LVCG than NGNM group as; 0.60±0.02 vs. 0.73±0.03, p<0, 04). E/A of tricuspid valve was lower in LVCG than NGNM group; E/A 0.52±0.07 vs. 0.67±0.05, p<0.01. E'/A' of tricuspid valve was also lower in LVCG than NGNM group; E/A 0.52±0.07 vs. 0.61±0.04, p<0.04. There were no significant differences between E/Aor E'/A' in NGNM group and controls; E/A of tricuspid valve 0.67±0.05 vs. 0.69±0.02 and E'/A' of tricuspid valve 0.61±0.04 vs. 0.63±0.06.

In multiple regression analysis, E'/A' of tricuspid valve independently correlated with E/A of mitral valve (r=0.76, p<0.01) and LVCG (r=0.78, p<0.03) but didn't correlate with LV mass. Furthermore, right ventricular wall thickness (RVWT) was 0.62±0.03 in LVCG group as compared to 0.36±0.08 in controls, (p<0.01) and RVWT independently correlated with septal wall thickness (SWT) r=0.56 p<0.001) and with left ventricle RWT, r=0.48, p<0.03, but not with LV mass or LVCG. The two groups LVCG and controls were similar for age; 67±16 vs. 66±03 years and for gender; 56.2% vs. 54.9% females respectively.

Discussion

The main finding of our study is a high prevalence of LVH, concentric geometry and LVDD in a population with RVDD. Biventricular diastolic dysfunction is a notable feature in these patients. RVDD associated with LVDD may be mediated by multiple mechanisms including increased after- load, elevated pulmonary venous and arterial pressures, partly as a protective mechanism against pulmonary edema [20,21]. Right ventricular performance and coronary perfusion may be adversely affected by altered LV hemodynamics, thereby suggesting the role of ventricular interdepedance [22] . This ventricular interdependence may also be due to common septal abnormality in the form of abnormally increased septal thickness, disruption of fiber orientation leading to septal dysfunction [23]. LV dilatation associated with LVH and LVDD in a limited pericardial compartment may also cause right ventricular diastolic dysfunction [24]. Despite the marked difference in muscle mass and chamber geometry, both ventricles are bound together by spiral muscle bundles that encircle the septum in a complex interlacing fashion to form a highly interdependent functional unit. Therefore, septal abnormalities may alter either left or right ventricular performance [25,26]. The components of septal architecture and the helical ventricular myocardial band may shed light on co-existence of biventricular dysfunction [27,28]. Distortion of this anatomic framework by of the left or right side of the heart can impair biventricular function and therefore septum is called the 'motor of biventricular function [23]. The septum is a midline structure between the two ventricles and it is composed of oblique fibers from two layers of the myocardial band; the descending and ascending segments of the apical loop [29] which result in septal thickness, whose weight accounts for approximately 40% of ventricular myocardial mass. As a consequence, patients with LVDD associated with RVDD exhibit greater impairment of LV morphology and function proportional to

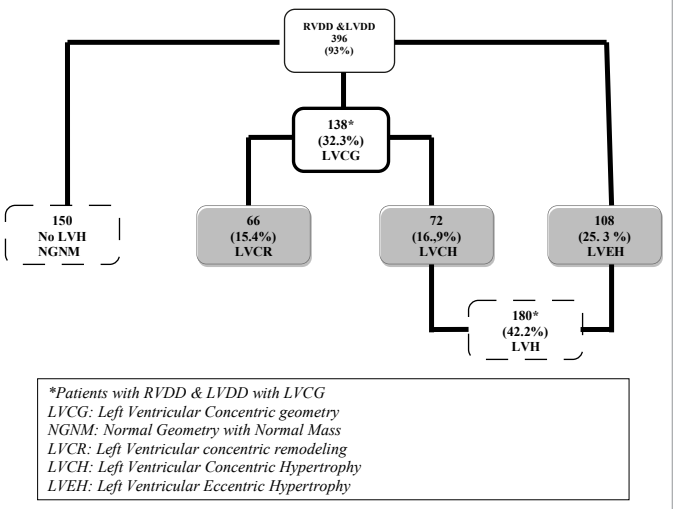

Figure 3: Patients divided according to LV geometry and mass.

the RV dysfunction compared with patients with LVDD alone [30,31]. A relatively load independent parameter, tricuspid TDI (E'/A') has been suggested to be a measure of global RVDD [32-34]. Among these patients with RVDD, we found a higher prevalence of LVCG which was independent of LVH. This suggests that it is concentric geometry rather than increased mass which was responsible for RVDD. The influence of LV mass on RV function has not been clearly delineated. Chakko and Habib did not find any relation between RV function and LV mass or LV wall thickness [29,30]. Our data are supported by Myslinski et al. [31], who reported that increased thickness of the inter-ventricular septum seems to be a major factor influencing RV diastolic function evaluated by right ventricular diastolic filling velocities**.** Our data showed that TDI of tricuspid valve (E'/A') was also associated with septal thickness. The septum is composed of oblique fibers surrounded externally by the right segment of the basal loop that contains predominantly transverse fibers in the intact heart. This obliquity of septal fiber orientation determines left and right sides of septum which contributes to LV systolic and diastolic function [35,36]. The septum also comprises of two muscle layers which works as a single unit contributing to biventricular geometry and function [37]. Concomitant LV and RV dysfunction may also be due to mechanical factors such as LV concentric geometry and increased septal thickness and coexisting bio-humoral factors as those derived by RAS system and others may also play role [38,39]. Our study didn't explore the correlation between RVDD and different stage of LVDD because of the smaller number of patients with advanced LVDD.

Conclusions

This study demonstrated that simple doppler trans-valvular inflow parameters and TDI may identify patients with biventricular diastolic dysfunction. This biventricular diastolic dysfunction was often seen in patients with LV concentric geometry, a pattern associated with an increased risk of cardiovascular events.

Acknowledgements

The authors thank the nurses Grazia Quaranta, Emmia Donativo, Maria Palazzo and Anna Paola Gennaro, for the support to the survey of the clinical data.

All authors have no financial disclosures, they report no biomedical financial interests or potential conflicts of interest.

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