Relationship of Ventricular Function and Morphometry in Patients with Symptomatic Severe Aortic Stenosis

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SUMMARY

Background
Ventricular hypertrophy is an adaptive mechanism of the myocardium to pressure overload in aortic stenosis. Different studies have postulated a correlation between structure and function in pressure overload due to aortic stenosis and the possible association with the development of pathological ventricular growth. However, there are a few studies evaluating these variables in hearts with compensated ventricular hypertrophy (without a significant increase in wall stress) and preserved ejection fraction.

Objective
To evaluate systolic and diastolic ventricular function in patients with symptomatic severe aortic stenosis with preserved ejection fraction and its correlation with collagen volume fraction and myocyte cross-sectional area.

Material and Methods
A total of 12 patients with symptomatic severe aortic stenosis were evaluated and compared with 6 patients without valvular heart disease; mean age was 65 ± 13 years and 58% were men. All patients underwent tissue Doppler imaging and cardiac catheterization. Endomyocardial biopsies were obtained to determine collagen volume fraction and myocyte cross-sectional area (μm2).

Results
Mean collagen volume was 6.1 ± 0.7%; mean myocyte cross-sectional area was 388.4 ± 15.8 μm2 and median strain in the basal septum was 14% (IIC 6.9-19). There was a significant correlation between septal strain measured by tissue Doppler imaging and collagen volume fraction (correlation coefficient –0.79; p = 0.03). We found no correlation between septal strain and myocyte cross-sectional area (R2 = 0.15; p = 0.8). The max positive dP/dt normalized for left ventricular end-diastolic pressure obtained during cardiac catheterization had a negative correlation with the myocyte cross-sectional area (R –0.94; p = 0.005).The time constant of pressure decay (tau) increased by 55% ± 3,5% (p < 0,05) and had a positive correlation with the myocyte cross-sectional area (R = 0.81; p = 0.04).

Conclusions
This study demonstrates the presence of anomalies in diastolic and systolic function in patients with symptomatic severe aortic stenosis and preserved ejection fraction that correlate with structural changes in the left ventricle, represented by increased interstitial collagen volume fraction and myocyte cross-sectional area.

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Key words > Aortic stenosis - Ventricular function - Myocyte morphometry - Interstitial collagen

Abbreviations >

TDI	Tissue Doppler imaging	**HF**	Heart failure
SSAS	Severe symptomatic aortic stenosis	**LVMI**	Left ventricular mass index
PSWSI	Peak systolic wall stress index	**LVM**	Left ventricular mass
SF	Fractional shortening	**LVEDP**	Left ventricular end-diastolic pressure
MFS	Midwall fractional shortening	**LVSP**	Left ventricular systolic pressure
EF	Ejection fraction	**LV**	Left ventricle
LVH	Left ventricular hypertrophy		

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BACKGROUND

Valvular heart disease is still a major cause of morbidity and mortality. In the United States, valvular heart disease represents between 10% to 20% of all the cases of cardiovascular surgery and a high percentage of patients with cardiac conditions managed with medical treatment. Demographic data demonstrate that hospitalizations due to heart failure (HF) secondary to coronary artery disease are decreasing compared to those due to valvular heart disease. (1) We should remark that, excluding mitral regurgitation secondary to myocardial disease, aortic valve stenosis is the most common lethal valvular heart disease; (2) thus, it is extremely important to select the adequate timing for aortic valve replacement surgery.

Left ventricular hypertrophy (LVH) is the adaptive mechanism of the myocardium to pressure overload in aortic stenosis, representing a complex process of remodeling (3) and biological adaptation which results from increased wall stress and affects all the components of the myocardial wall. If the remodeling process continues, progressive worsening of cardiac function occurs. (4) Several papers have focused on studying the correlation between changes in left ventricular (LV) systolic and diastolic structure and function in aortic stenosis and its relation with progression to HF. (5-8) However, there are a few studies evaluating these variables in hearts with compensated LVH (without a significant increase in wall stress) and preserved ejection fraction (EF).

During the last years, novel echocardiographic techniques have been developed to study the function and structure of the ventricular wall and, thus, obtain information about left ventricular pump function, myocardial fiber function and contractile state. (9) Tissue Doppler imaging (TDI) gives thorough information about regional ventricular function by analyzing myocardial deformation (strain) and the change in strain over the change in time (strain rate). (10) These indices have demonstrated a significant correlation with the parameters of the contractile state.

The goal of this study was to evaluate systolic and diastolic ventricular function in patients with severe symptomatic aortic stenosis (SSAS) with preserved ejection fraction (EF) and to correlate the functional findings with the structural changes observed in biopsy samples.

MATERIAL AND METHODS

A total of 12 patients (age 65 ± 13 years, 58% were men) with SSAS confirmed by Doppler echocardiography (aortic valve area < 1 cm2) with LVH (defined by LV wall thickness > 2 SD adjusted for age and body surface area) and EF > 50% were prospectively included. The control group consisted of 6 patients undergoing cardiac catheterization without evidence of coronary artery disease and normal Doppler echocardiography. Histopathological studies were performed in 6 subjects with no history of known cardiovascular diseases who suffered traumatic death.

Patients with cardiomyopathies were excluded from the study. All patients gave their consent to be included in the study and signed an informed consent form according to the Committee on Ethics of the Hospital Universitario Austral.

Echocardiography study

Doppler echocardiography and TDI were performed using a Vingmed System FiVe ultrasound scanner (GE Vingmed, Horten, Norway) with 2.5 to 3.5 MHz transducers. Six participants (age 53 ± 12 years) with no signs of LVH or coronary artery disease were included as echocardiographic control group.

All measurements were performed following the recommendations of the American Society of Echocardiography. The analysis of the variables was performed by two independent reviewers. (11)

Fractional shortening was calculated as: (LVEDD - LVESD) / LVEDD × 100 (LVEDD: LV end-diastolic dimension, LVESD: LV end-systolic dimension). Left ventricular volume and ejection fraction were calculated with the Simpson's rule using the formula: (LVEDV - LVESV/LVEDV) × 100 (LVEDV : LV end-diastolic volume, LVESV: LV end-systolic volume). (12) Midwall fractional shortening (MFS) was also estimated. (13)

Regional systolic function was evaluated using two-dimensional TDI (frame rate > 100) with off line pulsed TDI. Systolic velocity (S wave), presistolic velocity (s' wave), rapid filling velocity (tissue Doppler e wave) and myocardial velocity associated with atrial contraction (tissue Doppler a wave) were measured and expressed in cm/sec. Two dimensional strain was used to evaluate systolic strain obtained at the basal segment of the interventricular septum. The region of interest (ROI) was manually adjusted and the negative peak of the curve was identified after the QRS complex. Intraobserver and interobserver variability of strain measurements were analyzed with mean percent error (8% and 8.6%, respectively). Left ventricular mass (LVM) was determined by Devereux's and Reichek's formula and corrected for body surface area (g/m2) (left ventricular mass index [LVMI]). Mass (g) = 1.04 [(IVSTD + PWTD + LVDD)3 – LVDD3)] – 13.6 (IVSTD: interventricular septal thickness in diastole; PWTD: posterior wall thickness in diastole). (14)

Peak systolic wall stress index (PSWSI) in mm Hg was calculated as SBP + peak gradient × (LVDD / 2) / PWTD (SPB: systolic blood pressure, LVDD: left ventricular diastolic dimension, PWTD: posterior wall thickness in diastole). (15)

Images of tissue characterization were obtained at a frame rate > 100 frames/second and a sound intensity range of –80 - 0dB. Integrated backscatter and its cyclic variations were determined by a semi-automatic adjustment of the region of interest.

Cardiac catheterization

All patients underwent cardiac catheterization before surgery. Six subjects (age 53 ± 12 years) with no signs of LVH or coronary artery disease were included as control group.

The femoral artery and the femoral vein were punctured. A Swan-Ganz catheter was advanced and positioned at the level of the pulmonary artery. Pressures were measured and cardiac output was determined using the thermodilution method. A preshaped left coronary artery catheter was advanced and angiography of the left coronary artery was performed. The catheter was removed and another preshaped right coronary artery catheter was introduced for angiography or the right coronary artery. Finally, LV pressure and transvalvular aortic gradient were measured and recorded in a Philips XPER (XIM) Xper Information System (XIMs), Version 1.2.0.1474 connected to an Edwards Life Science transducer. The following variables were

measured: pressure in the aorta, LV systolic pressure (LVSP, mm Hg) and LV end-diastolic pressure (LVEDP, mm Hg). Maximum rate of rise of left ventricular pressure (+dP/dtmax, mm Hg/sec) was estimated. The time constant of the LV pressure decay during the isovolumic relaxation period was calculated as the negative inverse slope of the natural log of the pressure versus time relationship (lin tau, msec) and the time taken for LV pressure to fall to its 50% level (t50) from its initial level (–dP/dtmáx).

Determination of collagen and morphometry

During aortic valve replacement surgery, samples from the anterior wall of the LV were obtained (1-2 mg). The samples were immediately fixed in PBS and buffered formalin and embedded in paraffin. The sections were stained with Picrosirius red to quantify interstitial collagen and with rhodamine to calculate the myocyte cross-sectional area. All the determinations were performed using an Olympus microscope, series CX 31TSF, connected to a digital camera to take pictures of the myocytes. Images were analyzed using software Image Pro Plus 6.0 and a power magnification of 400×. The samples obtained from autopsies of subjects who suffered traumatic death were used as controls.

Statistical Analysis

Discrete variables were expressed as frequencies and percentages. Continuous echocardiographic variables were expressed as median (M) and interquartile range (IQR). The remaining continuous hemodynamic variables were expressed as mean ± standard deviation (SD). Data were analyzed using the analysis of the variance. Spearman's rank correlation coefficient was used to determine the correlation between continuous variables. A p value < 0.05 was considered statistically significant.

RESULTS

Table 1 shows the echocardiographic characteristics of the population studied. Patients with SSAS presented a significant increase in LV wall thickness and LV mass, with a median of 199 g/m2 (IQR 166-268) and of 93 g/m2 (IQR 89-09) in controls ($p < 0.05$). Mean transvalvular aortic gradient measured by Doppler echocardiography was 52 mm Hg (IQR 42-67) and the aortic valve area was significantly reduced (M 0.8 cm2; IQR 0.6-1), suggestive of severe aortic stenosis.

Systolic ventricular function

Median EF and FS were 73% (IQR 68%-78%) and 35% (IQR 31%-40%), respectively, in the control group and72% (IQR 63-77%) and 43% (IQR 32%-46%), respectively, in patients with aortic stenosis (p = ns). Thus, LV pump function was preserved in patients with valvular heart disease.

Midwall fractional shortening (MFS) was 20% (M) (IQR 14.2-20.9%) in the control group and 22. 4% (M) (IQR 18.3-24.9%) in the group with aortic stenosis (p = ns).In addition, and in coincidence with the ejection parameters, M LV longitudinal strain estimated by TDI was 20% (IQR 18-23%) in the control group and 14% (IQR 6.9-19%) in patients with aortic stenosis ($p < 0.01$). In the analysis of systolic function by echocardiography, this was the only finding that correlated with structural changes which showed significant differences between both groups.

Table 2 shows the values of the hemodynamic variables. LV systolic pressure was 139 mm Hg (IQR 131-145) in the control group and 205 mm Hg (IQR 192-215) ($p < 0.05$) in patients with aortic stenosis. There were no significant differences in +dP/dtmax between both groups: 1585 (IQR 1466-2231) vs. 1959 (IQR 1283-2597) mm Hg/sec.

Diastolic ventricular function

There were no significant differences in LVEDP (see Table 2) and diastolic dimensions [47 mm (IQR 43-51 mm) vs. 49 mm (IQR 45-54)] between control and aortic stenosis groups. These findings are consistent with compensated hypertrophy with absence of dilation or myocardial stretching. However, patients with aortic stenosis presented greater ventricular relaxation time compared to controls (113 msec [IQR 95-120] vs. 86 msec (IRQ 80-96) , respectively, $p < 0.05$). These results coincide with the measurements of t50 and lin tau during cardiac catheterization, two indices that evaluate isovolumic relaxation. These variables increased in patients with aortic stenosis from 19.1 ± 1.9 and 24.1 ± 4 msec to 30.1 ± 2.4 and 43.1 ± 7.1 msec ($p < 0.05$), respectively (Figure 1). Doppler echocardiography parameters of transvalvular mitral flow (A-wave velocity and E/A ratio) and TDI parameters (e and a waves velocities, and e/a ratio) showed statistically significant differences between the control group and patients with aortic stenosis (see Table 1).

Determination of collagen and morphometry

Collagen volume fraction (Figure 2) was significantly higher in patients with SSAS (3.53 ± 0.1% vs. 6.1 ± 0.7%; $p < 0.05$). As expected, myocyte cross-sectional area (Figure 3) was significantly greater in patients with aortic stenosis (206.7 ± 15.8 μm2 vs. 388.4 ± 15.8 μm2; $p < 0.05$).

Correlation between structure and function

The correlation between the different variables of ventricular function, collagen volume fraction and myocyte cross-sectional area was evaluated. We have observed an inverse correlation between strain calculated by TDI and collagen volume fraction measured in the samples obtained from patients with aortic stenosis (correlation coefficient –0,79; p = 0.03) (Figure 4). In addition, the +dP/dtmax normalized for LVEDP (+dP/dtmax / LVEDP) had a negative correlation with the myocyte cross-sectional area (correlation coefficient : –0.94; p = 0.005). These findings suggest that changes in myocardial structure (fibrosis and hypertrophy) might reduce the contractile state, even if left ventricular pump function is preserved (Figure 5 A). These changes were not detected by MFS, a precise echocardiographic parameter for the evaluation of systolic function in the presence of LVH, which also had no correlation with the myocyte cross-sectional area, as opposed to the correlation described for the hemodynamic parameters.

The evaluation of diastolic function revealed that

t50 had a positive correlation with the myocyte cross-sectional area (correlation coefficient: 0.81; p = 0.04), suggesting that the impaired ventricular relaxation might be due to greater hypertrophy (Figure 5 B). In addition, we observed a negative correlation between LVEDP normalized for LV diastolic dimension and collagen volume fraction (correlation coefficient: 0.56; p = 0.048), implying abnormal ventricular stiffness. The changes in diastolic function detected by Doppler echocardiography had no correlation with the collagen volume fraction.

DISCUSSION

The present study demonstrates that patients with SSAS, compensated LVH and preserved pump function have systolic and diastolic abnormalities related with changes in the myocardial structure.

Adequate timing of surgical aortic valve replacement is extremely important during the progression of aortic valve stenosis, as the development of left ventricular dysfunction is the main complication of valvular heart diseases and, particularly, of aortic stenosis, increasing perioperative (16) and late postoperative mortality. (17)

The current guidelines recommend aortic valve replacement when the patient presents symptoms or left ventricular dysfunction expressed by an EF< 50%. (18) This cutoff point as expression of LV dysfunction has important limitations, as it considers only the EF as a parameter of ventricular function. Ejection fraction is an index of LV global systolic function that may be modified by several variables, particularly by LVH. (19) In this sense, Schaper et al. (5) studied patients with LVH due to aortic stenosis and its correlation with the myocardial structure, demonstrating a

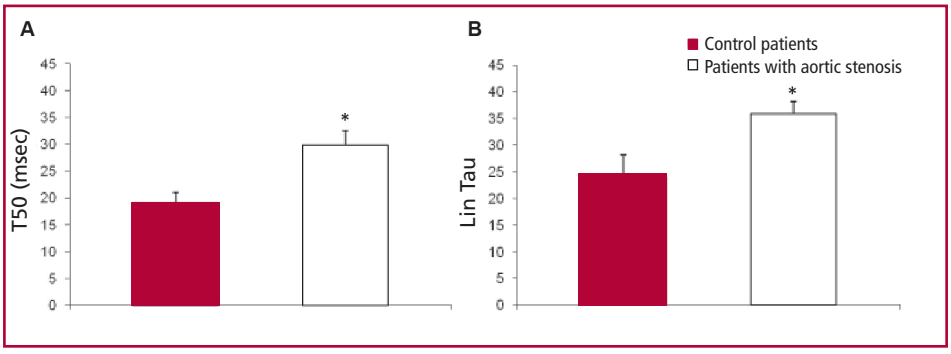

Fig. 1. Representation of the behavior of two indices of ventricular relaxation t50 (chart A) and linear tau (chart B). Both indices are significantly increased in patients with aortic stenosis, suggesting slow ventricular relaxation. * p < 0.05 vs. control.

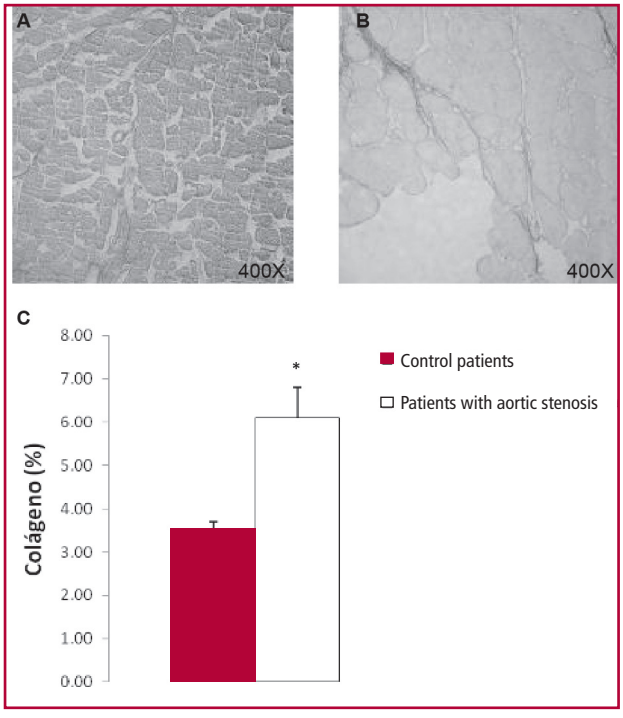

Fig. 2. Microphotographies of biopsy samples stained with Picrosirius red from controls (A) and from patients with aortic stenosis (B). The chart represents collagen volume fraction, which was significantly greater in patients with aortic stenosis. * p < 0.05 vs. control.

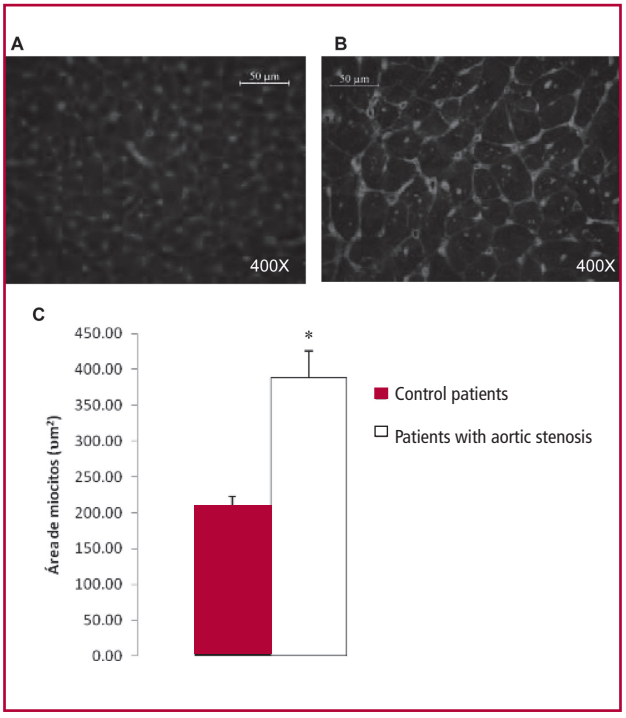

Fig. 3. Microphotographies of biopsy samples stained with rhodamine from controls (A) and from patients with aortic stenosis (B). The chart represents myocyte cross-sectional area, which was significantly greater in patients with aortic stenosis. * p < 0.05 vs. control.

Table 1. Echocardiographic variables

	Control (n = 6)		Aortic stenosis (n = 12)	
	Median	IQR	Median	IQR
Diastolic dimension (mm)	47	43-51	49	45-54
Systolic dimension (mm)	30	26-34	29	23-32
End-diastolic volume (ml)	99	77-106	102	74-110
End-systolic volume (ml)	29	18-38	28	16-37
Interventricular septum (mm)	9	8-9.75	15.3*	12.5-18
Posterior wall (mm)	8	7-9.75	12.5*	11-14,8
LV Mass (g/m2)	93	89-109	199*	166-268
Ejection fraction (%)	73	68-78	72	63-77
Fractional shortening (%)	35	31-40	43	32-46
Midwall fractional shortening (MFS)	20	14.2-20.9	22,4	18.3-24.9
Systolic stress (SS)	168	140-185	197*	170-312
Mitral E-wave velocity (cm/sec)	76	75-82	75	72-83
Mitral A-wave velocity (cm/sec)	60	54-60	112*	89-129
E/A ratio	1.1	0.8-1.2	0.66*	0.53-0.87
E-wave deceleration time (msec)	190	178-202	279*	246-304
Left ventricular relaxation time	86	80-96	113*	95-120
Tissue Doppler S-wave velocity of the basal septal wall (cm/sec)	5,5	4.7-6.1	3.5*	2.95-4.58
Tissue Doppler S-wave velocity of the middle septal wall (cm/sec)	3.8	3.1-4.3	2.66*	2.22-2.94
Basal-middle gradient in the septal segment by TDI	1.8	1.1-1.5	0.87*	0.46-1.43
Tissue Doppler s-wave in isovolumic contraction	2,1	0.75-3.4	2.25*	1.4-3.3
S/S' ratio	2.56	1.8-6.25	1.30*	0.92-2.71
Tissue Doppler E-wave velocity	7.8	5.6-9.8	4.27*	3.82-4.72
Tissue Doppler A-wave velocity	4.8	3.2-8.7	5.4*	3.92-5.9
Static backscattering (Db)	29	26-38	36*	32-40
Dynamic backscattering (Db)	7	6.25-7.9	7	6-7.25
Peak gradient (mm Hg)	8	4-10	89*	70-105
Mean gradient (mm Hg)	2.4	2-5	52*	42-67
Aortic valve area (cm2)	3.1	2.7-3.4	0.8*	0.62-1
Longitudinal strain measured by TDI (%)	20	18-23	14*	6.9-19

* $p < 0.05$ vs. control.

Table 1. Hemodynamic variables

	Control (n = 6)		Aortic stenosis (n = 12)	
	Median	IQR	Median	IQR
LV Systolic pressure (mm Hg)	139	131-145	205*	192-215
LV developed pressure (mm Hg)	124	121-130	189*	171-201
LV end-diastolic pressure (mm Hg)	14	9-16	15,6	11,1-22,9
+dP/dtmax (mm Hg/seg)	1585	1466-2231	1959	1283-2597
Systolic blood pressure (mm Hg)	142	126-147	147	125-147
Diastolic blood pressure (mm Hg)	74	67-77	77	63-94
Heart rate (bpm)	70	62-77	69	64-85
Transvalvular aortic gradient	3,8	3-4,1	53,7*	41-67

LV: Left ventricle. * $p < 0.05$ vs. control.

relationship between collagen content, myocyte degeneration and transition to HF. However, these authors subdivided the patients into different groups on the basis of EF. As we have previously mentioned, the evaluation of the contractile state in the presence of LVH is more precise when we use indices which analyze the behavior of the myocardial fiber. (19) The sensitivity and specificity of MFS is greater for the early detection of left ventricular dysfunction in the presence of LVH; yet this index was not abnormal in our study. We found that patients with aortic stenosis presented an abnormal +dP/dt normalized for preload parameters (LVEDP), an index more sensitive and specific to detect contractile failure. Other studies have performed strain analysis using Doppler echocardiography in patients with severe

aortic stenosis and preserved left ventricular ejection fraction (20) and reported abnormal values of this index without structural correlation. In our study we have measured strain using TDI for the evaluation of the systolic function, demonstrating a reduction in myocardial deformation (strain) compared to the control group and a correlation between this parameter and increased in collagen volume. These findings suggest that increased collagen volume fraction is an important component of myocardial deformation. In this sense, Weber el al. (21) reported that a two-fold increase in collagen volume is associated with LV diastolic dysfunction while a four-fold increase induces impairment of the systolic function. As we have previously mentioned, this impairment in systolic function could not be detected, even using MFS.

We have also shown the correlation between the degree of myocyte hypertrophy and +dP/dtmax / LVEDP, used as an index of contractile state. This finding strongly suggests that the pump function is preserved in these patients (EF > 50%), yet the contractile state is decreased due to hypertrophy.

From a physiopathological point of view, the progression to heart failure is based on changes on myocardial structure and function throughout the adaptive process of LVH. If the hypertrophic stimulus continues over time it will lead to deadaptation, as Meerson et al. (4) reported, becoming one of the mechanisms of permanent myocardial failure with its clinical expression: HF.

Diastolic function is another important aspect in the clinical evolution, functional class and outcomes of aortic stenosis. Diastolic changes appear early in the evolution of aortic stenosis and are directly responsible for the symptoms of the disease. Diastolic dysfunction is commonly associated with preserved systolic function and has been described in patients after (22, 23) and before aortic valve replacement surgery. Hess et al. (24) studied the correlation between diastolic abnormalities (24) with collagen volume and type and myocardial stiffness.

Isovolumic relaxation is another important aspect of the evaluation of diastolic function. These abnormalities have been widely studied in patients and in laboratory animals. (25) We have demonstrated a relationship between impaired relaxation and myocyte size: the greater the size of myocytes, the greater the impairment in ventricular relaxation.

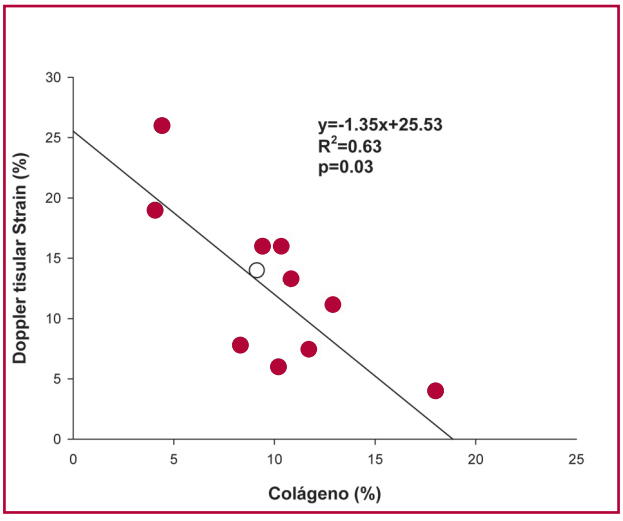

Fig. 4. Negative correlation between collagen volume fraction and strain determined by TDI. When collagen volume fraction increases, strain decreases. Correlation coefficient: –0.76; p = 0.049.

Study Limitations

Our study has some limitations. Firstly, it was performed on a small sample of 12 patients with aortic stenosis and 6 controls. Secondly, all the patients had symptoms, thus, the conclusions refer to this particular group. Interestingly, all these patients have preserved ejection fraction and normal left ventricular end-diastolic pressure, suggestive of compensated hypertrophy.

Thirdly, biopsy samples were obtained from the apex and the study of myocardial deformation was performed on the basal segments. Yet, we consider that this is a relative limitation, as hypertrophy has a uniform distribution and transmural biopsies gave information of all the myocardial layers.

Anatomopathological studies of the control group corresponded to subjects with traumatic death, without cardiovascular diseases, who underwent autopsies; endomyocardial biopsies were not performed in normal patients due to ethical reasons.

Finally, unfortunately 2D strain (speckle

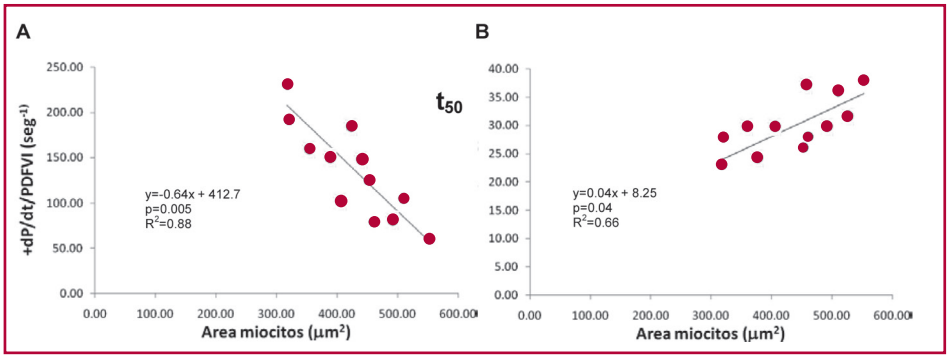

Fig. 1. A: Negative correlation between la +dP/dtmax/LVEDP and myocyte cross-sectional area (correlation coefficient : –0.94; p = 0.005). B: Positive correlation between t50 and myocyte cross-sectional area (correlation coefficient: 0.81; p = 0.04).

tracking), a method that allows the evaluation of all the myocardial segments, was not available at the moment of the study.

CONCLUSIONS

We have demonstrated that patients with SSAS, compensated LVH and preserved pump function have systolic and diastolic abnormalities related with changes in the myocardial structure (collagen and myocyte hypertrophy). The detection of these changes should be thoroughly investigated, as they might condition the evolution of patients with this disease and modify the surgical timing of patients with aortic stenosis and preserved LV indices in the future.

RESUMEN

Estudio de la función ventricular y su correlación con la morfometría en pacientes con estenosis aórtica grave sintomática

Introducción

En la estenosis aórtica, el mecanismo de adaptación miocárdica a la sobrecarga de presión es la hipertrofia ventricular. Diferentes trabajos han planteado la correlación entre estructura y función en la sobrecarga de presión por estenosis aórtica y su posible asociación con la evolución de la patología ventricular. Sin embargo, son escasos los trabajos en los que se evalúan estas variables en corazones con hipertrofia ventricular compensada (sin incremento significativo del estrés parietal) y con fracción de eyección conservada.

Objetivo

Evaluar la función ventricular sistólica y diastólica en pacientes con estenosis aórtica grave sintomática con fracción de eyección conservada y correlacionarla con el volumen de colágeno y el área miocitaria.

Material y métodos

Se estudiaron 12 pacientes, edad 65 ± 13 años, sexo masculino 58%, con estenosis aórtica grave sintomática y 6 pacientes sin patología valvular. En todos se realizaron Doppler tisular y cateterismo cardíaco; asimismo, se efectuaron biopsias intraoperatorias para determinar el volumen de colágeno y el área miocitaria (μm2).

Resultados

La media ± error estándar del volumen de colágeno fue del 6,1% ± 0,7%, la del área miocitaria fue de 388,4 ± 15,8μm2 y la mediana del strain tisular del septum basal fue del 14% (IIC 6,9-19). Se observó una correlación significativa entre el strain tisular del septum y el volumen de colágeno (coeficiente de correlación de –0,79; p = 0,03). No se observó correlación entre el strain tisular del septum y el área miocitaria (R2 = 0,15; p = 0,8). La +dP/dtmáx normalizada por presión de fin de diástole del ventrículo izquierdo obtenida en estudio hemodinámico se correlacionó en forma negativa con el área miocitaria (R –0,94; p = 0,005). La constante de caída de la presión (tau) se incrementó el 55% ± 3,5% (p < 0,05) y se correlacionó positivamente con el área miocitaria (R = 0,81; p = 0,04).

Conclusiones

El presente trabajo demuestra que en los pacientes con estenosis aórtica grave sintomática y fracción de eyección conservada existen alteraciones de la función sistólica y diastólica que se correlacionan con cambios estructurales del ventrículo izquierdo, representados por un incremento del volumen de colágeno intersticial y del área miocitaria.

Palabras clave > Estenosis de la válvula aórtica - Función ventricular - Morfometría miocitaria - Intersticio y colágeno

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