Short Communication

Left Atrial Systolic Function in Primary and Familial Amyloidosis: Assessment from Left Atrial Volume Change

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Summary: The severity of left ventricular involvement may differ between primary (PA) and familial amyloidosis (FA). This study examined whether differences in left atrial (LA) systolic function are also present. Twenty-eight patients (18 men, 10 women, aged 59 ± 12 years) with PA, 17 (11 men, 6 women, aged 40 ± 11 years) with FA, and 25 normal controls (18 men, 7 women, aged 56 ± 14 years) underwent transthoracic M-mode, two-dimensional, and Doppler echocardiography. Left atrial volumes were determined at mitral valve (MV) opening (maximal, $V_{\max}$), electrocardiographic P wave (onset of atrial systole, V_p), and MV closure (minimal, $V_{\min}$) from the apical two- and four-chamber views using the biplane area-length method. Left atrial systolic function was assessed with the LA active emptying volume (ACTEV) = $V_p - V_{\min}$ and fraction (ACTEF) = ACTEV/ V_p . The E/A ratio was increased (1.34 ± 0.93 vs. 0.89 ± 0.3), whereas deceleration time was decreased (168.1 ± 33.7 vs. 196.2 ± 34.2 ms) in PA compared with FA ($p < 0.05$). $V_{\max}$ and V_p were similar in PA and FA and greater than in the controls (46.6 ± 14 vs. 40 ± 11.4 vs. 27.1 ± 6.3 cm³/m², $p < 0.01$, and 33.4 ± 11.6 vs. 29.7 ± 10.8 vs. 16.8 ± 3.8 cm³/m², $p < 0.01$, respectively). The ACTEV was lower in PA and in the controls than in FA (6.7 ± 2 vs. 6.2 ± 2.2 vs. 8.5 ± 3.3 , respectively, $p < 0.05$). The ACTEF was lower in

PA than in FA and both were lower than those in the controls ($20 \pm 5\%$ vs. $28 \pm 7\%$ vs. $36 \pm 11\%$, respectively, $p < 0.01$). Despite a similar increase in LA volume, LA systolic dysfunction is more pronounced in PA than in FA. This is most likely due to the restrictive left ventricular physiology possibly associated with depressed LA contractility in the former.

Key words: left atrial systolic function, amyloidosis

Introduction

Cardiac amyloidosis is characterized by protein deposition in the myocardial interstitium, which leads to a “stiff heart” with relaxation abnormalities in the early and systolic dysfunction in the advanced stages.^{1–3} In a previous study we demonstrated significant differences between primary and familial amyloidosis regarding left ventricular (LV) systolic and diastolic function.⁴ Moreover, as amyloid fibrils may invade any part of the heart, infiltration of the atria may lead to depressed atrial contractility.^{1,5} The purpose of this study, therefore, was to evaluate left atrial (LA) mechanical function in patients with primary and familial cardiac amyloidosis using volume-derived indices of LA function.

Methods

Patient Population

Over a period of 10 years (1985–1995), 28 consecutive patients with primary and 17 with familial amyloidosis were studied; 25 normal subjects served as the control group. The study protocol was approved by the institutional committee on human research, and informed consent was obtained. Recruitment of all patients followed referral for symptoms or for abnormal electrocardiographic (ECG) or echocardiographic findings.

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The diagnosis of amyloidosis was confirmed by biopsies of subcutaneous fat, rectum, kidney, bone marrow, gum, or sural nerve. The disease was classified as primary when it was associated with signs of clonal plasma cell dyscrasia (light chain in serum or urine detected by immunofixation and tissue immuno-characterization of the amyloid protein) and familial when there was a positive family history, tissue immunocharacterization of the amyloid protein, and absence of light chains in serum and urine.⁶

The diagnosis of cardiac involvement was based on typical echocardiographic features (thickening of the cardiac walls, valves, and papillary muscles, with characteristic granular sparkling, biatrial enlargement, and decreased systolic thickening associated with diastolic and frequently systolic dysfunction) and ECG findings (low-voltage complexes, pseudoinfarction pattern complexes, rhythm and conduction disturbances).^{7–10} Patients with arterial hypertension, hypertrophic cardiomyopathy, or senile amyloidosis were excluded from the study.

Echocardiography

Comprehensive echocardiographic examination with pulsed, continuous, and color Doppler was performed with a Sonolayer SSH 160A ultrasound system (Toshiba, Crawley, West Sussex, UK), using 2.5 or 3.75 MHz transducers. From the two-dimensional guided M-mode echocardiogram, LV end-systolic and end-diastolic dimensions, interventricular septum (IVS), and LV posterior wall thickness (PWLTV) were measured.¹¹ Left atrial volumes were measured at mitral valve (MV) opening (maximal), onset of atrial systole (P wave of the simultaneously recorded ECG), and at mitral valve closure (minimal) from the apical two- and four-chamber views, as described previously.^{12–14} All values were obtained as the mean of three consecutive beats and were corrected for body surface area. Left atrial systolic (active emptying) function was assessed by use of the following parameters:^{15–18} (1) Left

atrial active emptying volume (ACTEV): left atrial volume at onset of atrial systole minus left atrial minimal volume; and (2) left atrial active emptying fraction (ACTEF): left atrial active emptying volume divided by the left atrial volume at onset of atrial systole.

The transmitral flow velocity profile was assessed with pulsed Doppler echocardiography. The following parameters were calculated: peak early velocity (E wave), peak velocity at the time of atrial contraction (A wave), E/A ratio, deceleration time of the peak early velocity (DT), and the isovolumic relaxation time (IVRT) with simultaneous recording of LV inflow and outflow velocities.^{19,20} The transmitral flow velocity pattern was considered restrictive based on the following criteria: (1) increased E wave, (2) decreased A wave with E/A ratio ≥ 2 , and (3) deceleration time ≤ 150 ms.²¹

Statistical Analysis

Values are expressed as mean $\pm$ standard deviation. Inter-group comparisons of continuous variables were performed using analysis of variance and the Scheffe test, whereas inter-group comparisons of discrete variables were performed using contingency tables. A *p* value of < 0.05 was considered statistically significant.

Results

The demographic and clinical characteristics of the study population are summarized in Table I. Patients with familial amyloidosis were significantly younger than those with primary amyloidosis and the controls. Moreover, the prevalence of New York Heart Association (NYHA) class III or IV was significantly higher in primary than in familial amyloidosis. Gender distribution, heart rate, and systolic and diastolic blood pressures were not significantly different in the three groups.

TABLE I Demographic and clinical characteristics of the study population

Characteristic	Primary amyloidosis (n = 28)	Familial amyloidosis (n = 17)	Control (n = 25)
Age (years)	59 $\pm$ 12	40 $\pm$ 11 ^a	56 $\pm$ 14
Sex (male/female)	18/10	11/6	18/7
Heart rate (beats/min)	86 $\pm$ 12	80 $\pm$ 15	82 $\pm$ 11
Systolic blood pressure (mmHg)	110 $\pm$ 9	112 $\pm$ 16	124 $\pm$ 20
Diastolic blood pressure (mmHg)	74 $\pm$ 10	73 $\pm$ 13	78 $\pm$ 9
CHF - NYHA Class			
I (%)	3 (11)	10 (59) ^a	—
II (%)	6 (21)	5 (29)	—
III (%)	14 (50) ^b	2 (12)	—
IV (%)	5 (18) ^b	0 (0)	—

^a *p* < 0.05 vs. primary amyloidosis or control.

^b *p* < 0.05 vs. familial amyloidosis or control.

Abbreviations: CHF = congestive heart failure, NYHA = New York Heart Association.

The parameters of LA function and transmitral diastolic flow are summarized in Table II. Specifically, the LA maximal volume was similar in primary and familial amyloidosis and greater than in the controls. The LA ACTEV in primary amyloidosis and controls was lower than in familial amyloidosis, whereas the LA ACTEF was depressed in both forms of amyloidosis compared with controls and was lower in the primary than in the familial form. The intra- and interobserver variability for LA volume measurements was 5.4 and 5.7%, respectively.

The ratio of peak early to late diastolic transmitral flow velocity was similar in primary amyloidosis and the controls and greater than that in familial amyloidosis. The LV deceleration time was lower in primary than in familial amyloidosis and the control group, whereas the LV IVRT was similar in the two forms of amyloidosis and greater than in the controls.

Discussion

The results of this study suggest that LA systolic function is significantly depressed in primary and moderately depressed in familial amyloidosis compared with controls.

Experimental and clinical studies have demonstrated that LA systolic function is affected by

(1) the precontraction fiber length, defining the maximum developed force according to the Frank Starling mechanism;^{22–24}

(2) the afterload, becoming a major determinant of LA ejection when the LA preload reaches its limit;²⁵

(3) the autonomic nervous system, which exhibits a positive inotropic effect via catecholamine release and a negative inotropic effect via the muscarinic receptors; and

(4) the rennin-angiotensin system, which exerts a positive inotropic effect via the AT1 receptors.^{26,27}

The mechanism of atrial systolic dysfunction in cardiac amyloidosis is incompletely understood, although amyloid infiltration of the atrial myocardium may result in a reduction in chamber contractility;⁵ however, other mechanisms may also be implicated. Specifically, Klein *et al.*²⁸ found that in most patients with cardiac amyloidosis and decreased A-wave velocity there are prominent atrial reversals in the pulmonary veins, arguing against a primary LA systolic failure as a mechanism for the impaired diastolic filling. Several studies^{29–31} have also shown that in patients with LV dysfunction the atrial systolic function correlated inversely with the pulmonary capillary wedge pressure, suggesting that the increments in LV diastolic pressure may have increased the atrial afterload and thereby limited atrial emptying. In addition, Sakai *et al.*³¹ propose that LA booster pump function improvement during dobutamine infusion seems to be induced by a reduced afterload on the left atrium due to improvement of LV compliance and not by changes of LA systolic shortening.

In the present study, despite the presence of LA dilation (increased preload), LA systolic function was depressed in both forms of amyloidosis and especially in the primary one. It is reasonable, therefore, to assume that this may be due to differences in LA contractility and/or LA afterload. Indeed, LA systolic and LV diastolic dysfunction resulting from myocardial immunoglobulin light chain infiltration, which occurs in the primary form, may be more severe than the corresponding atrial and ventricular dysfunction caused by prealbumin infiltration, which is a feature of familial amyloidosis.^{3,32} Moreover, the fact that the transmitral diastolic flow velocity profile was more frequently restrictive (25 vs. 11.8%) and the fractional shortening was lower in primary than in familial amy-

TABLE II Left atrial systolic function and transmitral flow of the study population

Parameters	Primary amyloidosis (PA)	Familial amyloidosis (FA)	Control
Left atrium			
Maximal volume Vmax (cm ³ /m ²)	46.6 ± 14 ^a	40 ± 11.4	27.1 ± 6.3
Volume at onset of atrial systole (cm ³ /m ²)	33.4 ± 11.6 ^a	29.7 ± 10.8	16.8 ± 3.7
Minimal volume (cm ³ /m ²)	26.7 ± 9.6 ^b	21.2 ± 7.5	10.8 ± 2.6
Active emptying volume (cm ³ /m ²)	6.7 ± 2 ^c	8.5 ± 3.3	6.2 ± 2.2
Active emptying fraction (%)	20 ± 5 ^d	28 ± 7	36 ± 11
Transmitral flow			
E/A	1.34 ± 0.93 ^e	0.89 ± 0.3	1.23 ± 0.46
Deceleration time (ms)	168.1 ± 33.7 ^f	196.2 ± 34.2	195 ± 27
Isovolumic relaxation time (ms)	99.7 ± 14.5 ^g	103.9 ± 7.8	80.2 ± 10

^a PA = FA > control, p < 0.01.

^b PA > FA > control (p < 0.01 PA & FA vs. control, p < 0.05 PA vs. FA).

^c PA = control < FA (p < 0.05).

^d PA < FA < control (p < 0.01 PA & FA vs. control, p < 0.01 PA vs. FA).

^e PA = control > FA, p < 0.05.

^f FA = control > PA, p < 0.05.

^g PA = FA > control, p < 0.01.

loidosis suggests that LV diastolic pressure and therefore LA afterload were most likely greater in the former.⁴

Study Limitations

The present study has the following limitations:

(1) Left atrial volume measurements were based on geometric assumptions, which may not have been thoroughly validated. Nevertheless, the biplane area-length method employed in this study has been used in patients with variable atrial size, and it has been recently suggested that the LA configuration approximates a cylindrical shape.^{12, 15, 16, 33}

(2) Patients with primary amyloidosis and controls were significantly older than patients with the familial type. However, aging is associated with an increased prevalence of abnormal LV relaxation, associated with LA dilation, preserved LA systolic function, and increased LA contribution to LV diastolic filling.^{30, 34} It is, therefore, highly unlikely that the observed differences in LA mechanical function between primary and familial amyloidosis are due to age difference between the two groups.

(3) Left ventricular diastolic pressure was not measured. However, the prevalence of a restrictive diastolic transmitral flow velocity profile was higher in primary than in familial amyloidosis, indicating that LV diastolic pressure and, therefore, left atrial afterload were most likely higher in the former.^{4, 35}

(4) Pulmonary vein flow was not assessed. Increased atrial reverse flow has been observed in cases with increased LV diastolic pressure. Thus, it is reasonable to assume that atrial reversals would be greater in primary than in familial amyloidosis and that this would result in an increase and not a decrease in LA active emptying fraction.^{3, 28, 35}

(5) A final limitation is the lack of endomyocardial biopsies in most of the patients in the present study. Nevertheless, those with biopsy-proven systemic amyloidosis and the typical echocardiographic features of cardiac involvement have confirmation by endomyocardial biopsy or autopsy.^{35, 36}

Conclusion

Left atrial dilation is a feature of both primary and familial amyloidosis. However, left atrial systolic dysfunction is more pronounced in the former and this may be due to depressed left atrial contractility and/or increased left atrial afterload.

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Letters to the Editor

Obesity Is Not Only an Adult Problem

C. R. Conti: *Clin Cardiol* 2004;27:183–184

To the Editor:

I share the same concern as Conti¹ that obesity is not only an adult problem. He cited the Bogalusa heart study involving a community, Bogalusa in Louisiana, of mixed (black and white) population. But the problem is not restricted to the Americans.

Childhood obesity is also increasing in prevalence in China.² In the most populous country in the world, where lean people used to be the norm,² this trend is alarming. In urban areas in China, the prevalence of childhood obesity increased from 1.5% in 1989 to 12.6% in 1997 and prevalence of overweight children increased from 14.6 to 28.9% in the same period.³ In a more recent study reported from China, the prevalence of overweight children was 27.7% in boys and 14.1% in girls.⁴

Although the cause of childhood obesity could be genetic, the major responsible factors are increased caloric intake and decreased physical activity. Thanks to the efficient advertisements of such fast food giants as McDonald's and Kentucky Fried Chicken, the Chinese children are devouring the Western fast food faster than ever.^{2, 5} On the basis of an 8-province survey, the fat intake of urban Chinese boys aged 12–15 years rose from about 17% of their total energy intake in 1989 to nearly 30% in 1993.⁶ On the other hand, increased physical inactivity also plays a role.⁷ The effect of television viewing on childhood obesity in China has been well documented: each hourly increment of television viewing is associated with a 1–2% increase in the prevalence of pediatric obesity in urban areas in China.⁸

Obese children grow up to be obese adults.² Therefore, to control obesity in the adults, we have to start to control obesity in the children. Conti cited evidence of fatty streaks in the blood vessels of aborted fetuses of mothers who smoked cigarettes during pregnancy.¹ These fetuses, of course, are helpless; but once they are born, it is up to them to do something to prevent themselves from getting fat.

Finally, as Conti said, parents need to be role models.¹ Because parents provide a child's contextual environment, they should be considered key players in interventions aimed at preventing children's weight-related problems.⁹

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