SCIENTIFIC LETTER

Left atrial remodelling after short duration atrial fibrillation in hypertrophic hearts

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Atrial fibrillation (AF) is the most common sustained cardiac arrhythmia found in hypertensive patients. AF causes atrial dilation, and progressive left atrial (LA) enlargement occurs once AF becomes chronic. In the Framingham heart study, LA enlargement was associated both with the duration of elevated blood pressure and with the level of systolic pressure.¹ The mechanism by which hypertension leads to LA enlargement seems to be related to haemodynamic changes. The LA size is closely related to diastolic filling pressure.

METHODS

We investigated 80 consecutive hypertensive patients with moderate left ventricular (LV) hypertrophy presenting with short duration AF (group A). The control population included 80 patients with lone AF and without cardiac hypertrophy (group B). The protocol was approved by the ethical committee and all patients signed an informed consent form. Patients underwent external DC shock. Cardiac evaluation included a complete echocardiogram. The following parameters were measured and compared. LA diameters were measured during systole along the parasternal long and short axis views. LA volumes were determined at mitral valve opening (maximal volume), at onset of atrial systole (p wave of ECG, P volume), and at mitral valve closure (minimal volume). LA volumes were measured from the apical four chamber and two chamber views by means of the biplane area length method and corrected for body surface area. LV internal dimension and wall thickness were measured in accordance with the American Society of Echocardiography guidelines. LV mass was normalised for body surface area to determine the left ventricular mass index (LVMI) (g/m²). Transmitral pulsed Doppler was recorded from apical four chamber view. Peak early filling (E), atrial filling (A) velocities, deceleration time, and pressure half time of E wave were measured. The atrial ejection force was calculated.² Effective mechanical atrial function was defined as the presence of atrial ejection force (AEF) > 7 dynes and of A wave peak velocity > 0.5 m/s.² The echo Doppler examination was performed one hour, one day, and seven days after the restoration of sinus rhythm.

Data are expressed as mean (SD). The significance of serial changes was determined by analysis of variance for repeated measures. Values for patients of different groups were compared using the Student unpaired *t* test.

RESULTS

Elective cardioversion for AF was accomplished with DC shock in all patients. The mean duration of arrhythmia was 40 (17) hours in group A and 44 (21) hours in group B (*p* = NS). Patients with LV hypertrophy had an increased LVMI 281 (46) compared to controls 112 (40) g/m² (*p* < 0.001). LA diameters and volumes were enlarged in all patients during AF (table 1).

Diastolic function was impaired in group A, the deceleration time of transmitral E wave was prolonged in group A at 282 (39) ms *v* 203 (34) ms in group B (*p* < 0.001), and the isovolumetric relaxation time (IVRT) was 112 (14) ms *v* 89 (13) ms.

Examinations performed one hour after the restoration of sinus rhythm showed a decrease of LA diameters and volumes in 43 (53.7%) patients of group A and in 68 (85%) patients of group B. The reduction of antero-posterior diameter was more notable than the reduction of supero-inferior diameter (−9% *v* −6% in group A and −7% *v* −3% in group B). Comparing the two groups, the reduction was more evident in group B compared to group A. The initial values of the peak A wave velocity and of the AEF in patients with LV hypertrophy were significantly lower compared to controls (peak A wave 53 (16) *v* 75 (14); *p* < 0.001 and AEF 6 (1.6) *v* 12 (2); *p* < 0.001). AEF was found to be > 7 dynes in 44 patients (55%) from group A compared to 67 patients in group B (83.7%; *p* < 0.01). An AEF > 7 dynes was associated with a more notable reduction in LA size. LA volumes were greater in hypertrophic patients who showed a delay in the recovery of atrial mechanical function.

At day 7 we reported six recurrences of AF in patients with a reduced atrial function and three recurrences in patients with a normal contraction.

Abbreviations: AEF, atrial ejection force; AF, atrial fibrillation; IVRT, isovolumetric relaxation time; LA, left atrial; LV, left ventricular; LVMI, left ventricular mass index.

Table 1 Serial evaluation of LA parameters in patients with LV hypertrophy

Parameters	Baseline	Post-cardioversion	Day 1	Day 7
LA antero-posterior diameter (mm)	44 (7)	40 (6)	40 (5)	37 (6)*
LA supero-inferior diameter (mm)	50 (6)	47 (4)	46 (5)	44 (6)*
Maximal volume (cm ³)	40 (7)	38 (6)	37 (6)	35 (5)*
P atrial volume (cm ³)		20 (5)	19 (5)	16 (6)*
Minimal volume (cm ³)	18 (2)	16 (3.1)	15 (2)	12 (4)*
AEF (dynes)		6 (1.6)	8 (2)	12 (3)
Peak A wave (cm/s)		53 (16)	60 (20)	64 (24)

**p* < 0.05.

At day 7, 67 patients (89%) with LV hypertrophy showed a good recovery of atrial mechanical function compared to 75 patients (94%) in group B.

DISCUSSION

The present study evaluated the effect of LV hypertrophy on the recovery of atrial mechanical function after cardioversion. LA diameters were found to be reduced after the recovery of sinus rhythm. Nevertheless we reported that a different rate of reduction and the presence of LV hypertrophy influences the magnitude of reduction. We found an interaction of LV hypertrophy and atrial mechanical and morphological remodelling. The delayed improvement in atrial contraction in patients with LV hypertrophy was associated with an initial stunning in atrial mechanical function and with a persistent dilation of the left atrium. The duration of AF seems to have a lesser influence on LA contractility with respect to the influence of LV hypertrophy. Regression of hypertrophy has been associated with reversal of the electrical remodelling in pressure overload states.³

The decrease in atrial myocyte contractility could be the cause of the decreased atrial function. The depressed atrial contractility is related to LV hypertrophy.⁴ Current evidence suggests that electrical remodelling depends upon the pattern of myocardial hypertrophy. We evaluated patients with concentric LV hypertrophy secondary to hypertension. All patients showed an abnormal relaxation pattern of diastolic dysfunction with prolonged IVRT and deceleration time. The LA stunning reported after cardioversion of AF predisposes patients to, and increases the short term risk of, thromboembolism.⁵ Patients with LV hypertrophy showed

two predisposing conditions to embolism: a pronounced LA mechanical stunning and a persistently dilated LA.

LV hypertrophy influences the recovery of LA function after cardioversion of AF. LA stunning is responsible for the prothrombotic state following cardioversion. Patients with LV hypertrophy had a higher risk of thromboembolism because of the slow recovery of LA stunning and a persistently dilated LA. These patients need effective anticoagulant treatment after cardioversion, even after AF of short duration.

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IMAGES IN CARDIOLOGY

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Percutaneous transluminal septal coil embolisation as an alternative to alcohol septal ablation for hypertrophic obstructive cardiomyopathy

Alcohol septal ablation appears to be a successful strategy for severe obstructive cardiomyopathy, resulting in reductions in clinical symptoms and left ventricular outflow tract gradient (LVOTG). However, rates of mortality (1–4%) and permanent complete atrioventricular (AV) blockage (15–30%) associated with alcohol ablation in experienced centres are similar to surgical myectomy. We proposed to perform coil septal embolisation in order to avoid alcohol toxicity. We report the case of a 43 year old patient, who presented with severe heart failure symptoms (New York Heart Association (NYHA) functional class III) refractory to medical treatment. Echocardiography showed a maximal 18 mm end diastolic thickness of the high septum, and a basal 75 mm Hg LVOTG. A coil was advanced and dropped into the first and second septal branches (see panel). The clinical follow up was event-free without ventricular arrhythmia and AV blockage. At three months, the patient felt a dramatic improvement in his dyspnoea (NYHA class I), and the LVOTG was 25 mm Hg. At five months, the patient remained asymptomatic. Further studies are warranted to evaluate this new strategy.

Coronary angiographic projection of the left anterior descending artery with first and second septal branches (arrows) before (A) and after (B) coil embolisation.

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