Left ventricular hypertrophy regression is persistent on antihypertensive therapy for 3 years.

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Background: The regression of left ventricular hypertrophy (LVH) has been shown following antihypertensive treatment, however the longterm persistence of such effect may be questionable.

The objective of this study was to assess the 3 year effect of rimelinenide (R) monotherapy on LVH in mild-moderate hypertension.

Methods: 45 consecutive patients were included into this prospective phase IV open echocardiography (echo) study who had baseline LVH defined as left ventricular mass index (LVMIV) =140 g/m2 and >150 g/m2 in females and males resp., in whom blood pressure was well controlled by 1-2 mg/day R monotherapy and who had measurements at baseline, at 1 year, at 2 years and 3 years. There were 20 males, 25 females, mean age 50±14.7 yrs. Echo measurements were performed by one "blinded" observer in a central laboratory. LV posterior wall (PW), septum (IVS) thickness, LV dimensions, E, A velocities, deceleration time (DT) were measured. LV mass index (LVMI), relative wall thickness (RWT), LV ejection fraction (EF), and E/A ratios were calculated.

Results: Baseline systolic function was normal (EF=56.8±4.7%). 59.1% had concentric hypertrophy and 59.5% had impaired relaxation (E/A<1 with DT>200 ms). There was no change in LV dimensions EF, E and DT. PW, IVS, LVMI and RWT decreased significantly at 1 year and these changes persisted after 3 years (Table). The frequency of concentric hypertrophy decreased from 59.1 to 24.4, 25.6 and 31%, the rate of abnormal RWT from 59.1 to 34, 42 and 36%.

Baseline 1 year p 2 years p 3 years p
PW mm 11.8±1.2 10.3±1.1 x x 10.5±1.2 NS 10.7±1.3 NS
IVS mm 12.2±1.7 10.5±1.2 NS 10.7±1.2 NS 10.7±1.3 NS
LVMI g/m2 162.1±10.1 32.9±2.3 NS 32.5±2.4 NS 173.5±29.4 NS
RWT% 48.1±9.3 41.9±6.2 NS 43.9±8.9 NS 43.3±6.4 NS

Conclusions: There was a significant regression of LVH after 1 year R monotherapy due to decrease in wall thickness with an improvement of LV remodelling and these changes persisted after 3 year monotherapy.

Peripheral endothelial dysfunction and left ventricular diastolic dysfunction in patients with essential hypertension.

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Similar neurohumoral factors are involved in myocardial and peripheral vascular endothelial dysfunction and impairment. However, it is not clear whether endothelial abnormalities are associated with left ventricular (LV) diastolic dysfunction.

The aim of the study was to investigate the relation of LV diastolic function parameters and plasma levels of soluble intercellular (s-ICAM) and vascular (s-VCAM) cell adhesion molecule and endothelium-dependent flow-mediated dilatation in brachial artery (FMD) in hypertensive pts.

Material and methods: Studied group consisted of 41 pts (18 males, 23 females) mean age 54±2.11.9 with essential hypertension and without coronary artery disease. 18 age-matched healthy persons served as controls. Echocardiographic assessment of LV diastolic function or s-ICAM and s-VCAM were noted.

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Determinants of exercise capacity in hypertensive patients.

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An impaired exercise capacity is common in hypertensive patients (pts). Not all determinants of this pathology remained exactly recognized.

The aim of the study was to investigate factors related to exercise tolerance in hypertensive pts.

Material and methods: Studied group consisted of 41 pts (18 males, 23 females) mean age 54±2.11.9 with essential hypertension and without coronary artery disease. In each patient echocardiographic study, estimation of plasma levels of ANP and BNP and treadmill exercise test were performed. Echocardiographic assessment comprised evaluation of left ventricular mass index (LVMI), ejection fraction (LVEF), velocity of early (E) and late (A) transmitral flow, deceleration time of E wave (DT), isovolumic relaxation time (IVRT), total ejection isovolume index (TEI), flow propagation velocity of E wave (Ep), velocity of systolic (S), diastolic (D) and atrial reversal (AR) pulmonary venous flow. Exercise capacity was assessed by exercise time and total workload expressed in MET.

In conclusion: Impaired exercise capacity is due to LVH since it was found in 25 pts (61%). Groups of pts with normal and impaired exercise tolerance did not differ with respect to age, LVMI, LVEF and ANP. Significantly higher values of A, S/D and BNP and lower values of D were noted in pts with diminished exercise capacity. Moreover, in this group of pts trends toward lower values of E/A and higher values of AR were observed. Significant correlations were found out for MET and: age (r=-0.49, p<0.001), A (r=-0.62, p<0.001), E/A ratio (r=0.55, p<0.004), D (r=0.55, p<0.004), AR (r=0.38, p<0.01), BNP (r=-0.53, p<0.001). Exercise time correlated with A (r=-0.61, p<0.001), E/A ratio (r=0.40, p<0.04), D (r=0.51, p<0.009), AR (r=0.35, p<0.02), S/D ratio (r=-0.47, p<0.01), BNP (r=-0.45, p<0.01). Other investigated parameters did not correlate with both MET and exercise time. By stepwise multiple linear regression analysis D and AR were the only determinants of MET whereas D and A turn out to be the only independent predictors of exercise time.

In conclusion: In hypertensive pts: (1) diastolic function of LV is a principle determinant of exercise capacity, (2) BNP is superior to ANP in predicting exercise tolerance.

Hypertrophic CMP

The localization of the septal ablation lesion is predicted by the septal contrast depot during echo-guided septal ablation.

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Background and Introduction: Percutaneous septal ablation (PTSMAS) for symptomatic, hypertrophic obstructive cardiomyopathy (HOCM) requires the exact definition of the septal myocardium to be attacked. We tested whether the clinical and haemodynamic effect is correlated with morphologic measures of the intra-procedural contrast study (p-MCE) in 33 patients (pts.) who had their echo video loops archived digitally and who had a complete follow-up after 3 months.

Results: The mean area of the contrast depot (CD) was 8.5±2.5 cm2, its length along the left ventricular (LV) endocardial border 1.9±0.6 cm, the proximal edge 1.0±0.3 cm upstream the mitral-septal contact (SAM-C), with the SAM-C covered region. The final shape of the ablation lesion and its hemodynamic effect, however, is predicted by the localization of the contrast depot with respect to the mitral-septal contact (SAM-C), with the SAM-C covered region. The final shape of the ablation lesion and its hemodynamic effect, however, are not correlated with measures of the contrast depot but seem to follow an individual remodeling process.