

100

Renin: a new therapeutic target in the prevention of cardiac remodeling in patients with abdominal obesity?

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Objectives: We investigated the association between aldosterone (ALDO) and renin (REN) plasma levels with early changes of cardiovascular structure and function involved in the progression to heart failure (HF) in subjects with abdominal obesity (AO).

Background: Incidence of HF doubles in AO. This condition is associated with an activation of the renin angiotensin aldosterone system, which may be involved in the progression to HF.

Methods: AO subjects matched for age and sex to healthy volunteers (HV) underwent ALDO and REN measurements and cardiac and arterial phenotyping to explore early remodeling (trans-thoracic echocardiography, cardiac magnetic resonance imagery, intima-media thickness measurement, pulse wave velocity, and fibrosis biomarkers).

Results: 116 AO subjects (BMI: 31.7 ± 3.4 kg/m²) aged 55 ± 6 years and 53 HV were recruited. AO subjects had higher ALDO (59 (33-106) vs 34 (18-65) pg/ml, $p < 0.0001$), left ventricular mass (LVM) (97 ± 25 vs. 84 ± 21 g, $p = 0.003$), and cardiac remodeling index (CRI=LVM/LV End Diastolic Volume) (0.69 ± 0.16 vs. 0.60 ± 0.10 g/ml, $p = 0.004$). AO subjects also had higher serum levels of the extracellular matrix turnover (PINP: 36 ± 16 vs. 22 ± 14 ng/ml, $p < 0.0001$; PIIINP: 2.6 ± 1.3 vs. 3.4 ± 6.7 ng/ml, $p = 0.082$; ICTP: 3.9 ± 1.0 vs. 4.6 ± 0.9 ng/ml, $p < 0.0001$). In the multivariate analysis REN was significantly correlated with LVM ($p = 0.036$) and CRI ($p = 0.009$). PIIINP ($p = 0.043$) was independently correlated with diastolic dysfunction.

Conclusions: In AO, there is an early rise in ALDO and early changes of extracellular matrix remodeling and cardiac geometry that were correlated to REN. Renin might be a target for the prevention of early cardiac remodeling and progression to HF in AO patients.

101

Long-term follow-up after myocardial contrast echocardiography-guided alcohol septal ablation for hypertrophic obstructive cardiomyopathy

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Aim: Report the immediate and long-term results of alcohol septal ablation (ASA) guided by myocardial contrast echocardiography (MCE) for treatment of symptomatic hypertrophic obstructive cardiomyopathy from a high-volume centre.

Methods and results: From 2000 to 2011, 159 patients (88 males) underwent ASA. Mean age was 56.4 ± 15.9 years (range 8-87). Medical treatment included beta-blockers (75%), calcium-channel antagonists (49%) and disopyramide (2%). 22% had prior pacemaker (PM) implantation, 2% prior implantable cardioverter-defibrillator (ICD) and 4% prior cardiac surgery. At baseline, mean New York Heart Association (NYHA) functional class was 2.8 ± 0.6 . Mean left ventricular outflow tract (LVOT) peak gradient and septal thickness were 92 ± 45 mmHg and 23.0 ± 3.8 mm, respectively. During ASA, 2.0 ± 0.8 ml of absolute alcohol was injected in 1.2 ± 0.4 septal perforators. Final procedural LVOT peak gradient was 20 ± 22 mmHg. Procedural success (immediate LVOT peak gradient reduction $> 50\%$) was achieved in 94%. Complications included coronary dissection requiring stent implantation (1 procedure). There were 2 in-hospital deaths (1 refractory ventricular arrhythmia, 1 complete atrioventricular block). In-hospital permanent PM implantation was required following 8.7% of procedures. One patient required

an ICD for non-sustained ventricular tachycardia. Mean peak CK was 932 ± 491 IU/L. At a mean follow-up of 2.3 ± 1.8 years after the procedure (range 0-8.4), there were 7 additional deaths (overall annual mortality of 2%). Repeat ASA was required in 11 patients (7%) and a new ICD was needed in 1 patient (5%). Mean NYHA class was improved to 1.2 ± 0.5 .

Conclusion: MCE-guided ASA is associated with a high rate of immediate success and a low rate of procedural complications. In addition, long-term follow-up shows sustained clinical benefit with a low rate of adverse events (annual mortality lower than the expected rate of 3-4% in this high-risk population).

102

Cardiovascular complications of acromegaly

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Cardiovascular disease in acromegaly is multiple pathogenic mechanisms (high blood pressure (hypertension), valve disease, coronary atherosclerosis, diabetes ...) but also by a proper action of human growth hormone (GH) on the myocardium. The acromegalic cardiomyopathy is the most common, it is the leading cause of mortality in acromegaly.

Purpose, Materials and Methods: We propose in this retrospective study to report on the cardio-vascular clinical profile, echo-cardiographic (size cavities, cardiac mass, valvular thickening, valvular leakage, systolic and diastolic function) and changing (before and after treatment and medical or surgical) of 20 acromegalic patients followed concurrently with doctors endocrinologists.

Results: The population is predominantly male (70%) whose mean age was 47.21 ± 15.81 (range: 27-67 years) 30% of patients under 40 years 40% of patients were hypertensive and 50% have diabetes. The diagnosis of acromegaly is sometimes late compared to early clinical signs: 9.9 ± 8.6 years (range 1-30 years).

Conclusion: Morbidity and mortality associated with acromegaly is considered equivalent to the general population according to recent studies

Results

	Before treatment	After treatment
CLINICAL FEATURES		
Dyspnea (NYHA stage)	II (4 pts)	II (2 pts)
Congestive Heart	0	0
LVH	13 (65%)	9 (45%)
Arrhythmia / conduction	0	0
Repolarization disorder	1	1
MI failure	1	1
Hypertension	8 (40%)	5 (25%)
Diabetes	10 (50%)	8 (40%)
SONOGRAPHIC FEATURES		
EF (%)	70.5 ± 8.3	72.1 ± 10.3
Septal thickness (mm)	12.04 ± 2.8	11.01 ± 4.9
Post Wall Thickness (mm)	12.04 ± 2.8	10.11 ± 4.2
Myoc Masse (g/m ²)	167 ± 42	138 ± 63
Thickening M/Ao =9	3/6	3/6
E/E'	13.09 ± 4.9	8.2 ± 3.7
E deceleration time	271 ± 46	199 ± 38
PAPS	34.9 ± 4.9	34.9 ± 4.9
Mitral regurgitation (grade 1)	I (1/3)	I
Ao regurgitation (grade 2)	I (1/6) II (1/6)	I et II
Cardiac output	7.3 ± 1.9	5.5 ± 4.2