Clinical Cardiac Function and Heart Failure

132A ABSTRACTS - Cardiac Function and Heart Failure

1015-160 Quantification of Myocardial Injury After Percutaneous Transluminal Septal Myocardial Ablation in Hypertrophic Obstructive Cardiomyopathy

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Background: Percutaneous transluminal septal myocardial ablation (PTSMA) is a non-surgical therapeutic procedure for reducing left ventricular outflow tract obstruction in symptomatic patients with hypertrophic obstructive cardiomyopathy (HOCM). The aim of this study was to evaluate septal myocardial injury after PTSMA using delayed contrast-enhanced (DE) cardiac magnetic resonance imaging (CMR) and to compare the results with those of patients undergoing myectomy (MYE). Methods: 12 patients (mean age 52±15 years, 7 males) underwent MIR before and 4 weeks after PTSMA; volume of ethanol injected during procedure was 1 to 5 mL. Images were acquired on a 1.5 T scanner (Vision/Sonata, Siemens, Erlangen, Germany). Cine steady-state free precession (SSFP) images (TE 2.6 ms, TR 7.6 ms, TI 260-300 ms) were acquired at follow up, 20 to 30 minutes after i.v. administration of 0.2 mmol/kg gadolinium-DTPA. Left ventricular function parameters, myocardial mass, and hyperenhanced area's (including central dark zones of hypoenhancement) were quantified using the MASS software package (Leiden University Medical Center, the Netherlands).

Results: Left ventricular mass values before and after PTSMA were 235.6±70.7 g vs. 225.2±71.7 g (p=0.001), respectively. Septal myocardial mass pre- and post-PTSM was 73.2±29.4 g vs. 72.7±26.2 g, resp. In all patients the injured myocardium was well visualized. The hyperenhanced septal myocardial mass was in the range of 3.6–24.9 g [mean: 12.8±7.8 g], and involved 5.0±4.0 % of the post-ablational total LV mass vs. 2.1±1.2 % of the septal myocardial mass. Myocardial injury size was not correlated with the volume of ethanol administered. Conclusions: The extent of myocardial injury after PTSMA can be determined using DE-CMR and was not correlated with the volume of ethanol administered.

1015-161 Untoward Effect of Septal Reduction Interventions on Conduction System in Patients With Obstructive Hypertrophic Cardiomyopathy

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Background: Previous studies have found that percutaneous septal reduction interventions (PSRI) may cause myocardial dysfunction and conduction abnormalities. We therefore evaluated conduction abnormalities after percutaneous transluminal septal myocardial ablation (PTSMA) and myectomy (MYE) procedures. The aim of the study was to observe the impact of both procedures on the conduction system. Hypertrophic Cardiomyopathy (HCM) patients may develop conduction abnormalities after percutaneous transluminal septal myocardial ablation (PTSMA) and left branch bundle block was often caused by MYE. Severe AV block may develop when both procedures are required.

Methods: Pts with resting LVOTO who have poorer indices of cardiac function/exercise capacity. Non-obstructive HCM pts with exercise-induced LVOTO have cardiomyopathic indices consistent with better stroke volume augmentation during exercise.

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Conclusions: PTSMA and MYE significantly reduced LVOT PG. However, right branch bundle block was frequently produced by PTSMA and left branch bundle block was often caused by MYE. Severe AV block may develop when both procedures are required.

1016-162 Prediction of Sudden Death in Patients With Dilated Cardiomyopathy Receiving Angiotensin Converting Enzyme Inhibitors and Beta-Blockers: The Extent of Interstitial Myocardial Fibrosis and the Presence of Nonobstructed Ventricular Tachycardia


Background: Recent clinical trials have shown that angiotensin-converting enzyme inhibitors (ACEI) and beta-blockers (BB) reduce the mortality in patients with dilated cardiomyopathy (DCM). However, some DCM patients still died suddenly in spite of the administration of ACEI and BB. We tried to identify patients at risk for sudden death in DCM patients receiving ACEI and BB. Methods: We studied 50 DCM patients (NYHA class: 2.6±0.6, left ventricular ejection fraction: 32±8%) receiving ACEI (78%) and BB (76%). At the baseline, we performed echocardiography, 24 h ECG monitoring, right side cardiac catheterization, left ventricular endomyocardial biopsy. The extent of myocardial fibrosis was estimated by the point count method in all biopsy samples. Moreover, after classifying myocardial fibrosis sequentially into two types, interstitial (I) and interfascicular fibrosis, by the distribution of fibrosis on each cross point, we also estimated the extent of each fibrosis.

Results: During the observation period of 58±28 months, 8 patients died suddenly. By multivariate Cox analysis, out of the variables including clinical, hemodynamic, echocardiographic parameters, the presence of nonobstructed ventricular tachycardia (VT: more than 5 consecutive ventricular ectopic beats) and the extent of myocardial fibrosis, the presence of nonobstructed ventricular tachycardia (VT) with a higher extent of I fibrosis was strongly and independently associated with sudden death. Kaplan-Meier analysis revealed that patients with a greater extent of I fibrosis (>12.5%) and nonobstructed VT (Group I) showed significantly higher rate of sudden death than patients with a lesser extent of I fibrosis or the absence of nonobstructed VT (Group II) (67% vs 9%, p<0.001).

Conclusion: The extent of I fibrosis and the presence of nonobstructed VT would be a powerful predictive marker for sudden death in DCM patients receiving ACEI and BB.

1016-163 QT Dispersion is Not Associated With Left Ventricular Function in Elderly Patients With Symptomatic Heart Failure: ELITE II Substudy

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Background: It remains controversial if QT dispersion (QTd) is associated with left ventricular function and the prognostic value of QT in HF secondary either to idiopathic dilated cardiomyopathy (ICD) or ischaemic heart disease (IHD), we studied 966 HF pts (age 71±7 years, 703 men) enrolled into the ELITE II trial. Of study pts, 766 had IHD (age 71±7 years, 573 men) and 105 had ICD (age 71±7 years, 65 men). QT intervals were manually measured on standard 12-lead ECGs using an in-house computer assisted system. Results in all study pts, the mean QTd and JTD were 46±15 ms and 83±32 ms, respectively. In pts with LVEF<25%, heart rate was significantly higher (p<0.001) and QT dispersion was significantly shorter (25% (95±15 vs 95±13 ms). No significant difference in QTd was found between pts with and without NYHA class III/IV (87±31 vs 84±32 ms). QTd was not correlated with LVEF (<0.004) or NYHA (r=0.04). During follow-up (540±153 days), 119 (12%) pts died of cardiac cause (CD), including 59 (6%) sudden death (SD). A prospective cut-off value of cardiac death (CD) or SD was identified with CD or SD, while significantly more patients with LVEF<25% died of CD than those with LVEF>25% (16% vs 11%, p=0.045). There were no significant differences in age and NYHA functional class between IHD and IDC. More male (75% vs 62%, p=0.006) and higher LVEF (31±6.8 vs 28±2.7%, p<0.001) were found in IHD than IDC. The mean QTd in IHD were similar to IDC (86±32 vs 87±27 ms) and it was not related to mortality at all. The frequency of CD or SCD were not significantly different in IDC and IHD (11%, 5% vs 13%, 6%, respectively, p=NS). In both IHD and IDC, a QT<420 ms failed to predict CD or SCD. The results remains unchanged when LVEF was analysed in the same manner.

Conclusions in this ELITE II substudy, QTd (and JTD) was neither associated with left ventricular function, nor with aetiology of heart failure. Reduced LVEF remains a significant predictor of CD but increased QTd did not predict mortality irrespective of aetiology of HF.