

RESULTS There were no differences in the occurrence of major adverse cardiovascular events at 30 days and 1 year among three groups. There was significant improvement in the change of end-diastolic volume (LVEDV) in HP-BMC group compared with N-BMC or control at 12 months ($P < 0.05$). No differences were observed in the change of left ventricular ejection fraction (LVEF), left ventricular end-systolic volume (LVESV) or wall motion score index (WMSI) among three groups. Nonetheless LVESV were decreased in the HP-BMCs group compared with baseline ($P < 0.05$, within group), but not the N-BMCs or control group. WMSI was improved in HP-BMCs and N-BMCs group ($P < 0.05$, within group), but not in control. The ratio of myocardial perfusion defect determined by SPECT decreased in HP-BMCs and N-BMCs groups ($P < 0.05$, within group), but no significant differences were observed among three groups.

CONCLUSIONS Our results provide the first-in-man evidence that intracoronary administration of HP-BMCs following acute MI appears to be safe and feasible. These results provide the basis for future prospective randomized clinical trials in a larger patient cohort.

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Circulating miR-499 are novel and sensitive biomarker of acute myocardial infarction

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OBJECTIVES The aim of this study was to investigate the value of plasma microRNA-499 (miR-499) as a novel biomarker for early diagnosis of acute myocardial infarction (AMI).

METHODS Enrolled in this study were 227 patients with chest pain on presentation to the departments of emergency and cardiology of Wuxi Second People's Hospital between October 2011 and May 2014. Additional 100 healthy individuals who received physical examination in the same hospital during the same period were used as control. Plasma was collected at admission, and the abundance of miR-499 was measured using reverse transcriptase-polymerase chain reaction (RT-PCR).

RESULTS MiR-499 was significantly elevated in 142 patients diagnosed with AMI as compared with 85 patients in non-AMI group and 100 subjects in healthy control group. Plasma miR-499 were already detectable in the plasma as early as 1 h after onset of chest pain in AMI patients, and continued to increase gradually without any sign of decreasing tendency within 9 h in AMI patients. miR-499 was highly positively correlated with the serum creatine kinase-MB (CK-MB) and cTnI. The area under the curve (AUC) of miR-499 for the diagnosis of AMI was 0.86, with an optimal cut-off value of 4.79, sensitivity of 80%, and specificity of 80.28%.

CONCLUSIONS miR-499 was shown to substantially increase the diagnostic accuracy of CK-MB and cTnI in the diagnosis of AMI, and therefore it may prove to be a useful marker for early diagnosis of AMI.

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Xanthine oxidase inhibition prevents atrial remodeling and atrial fibrillation in alloxan-induced diabetic rabbits

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OBJECTIVES Accumulating evidence suggests that xanthine oxidase (XO) activation is one of the major sources of reactive oxygen species (ROS) and may play an important role in the initiation and perpetuation of atrial fibrillation (AF). XO inhibitor allopurinol may exert beneficial effects on diabetes-related atrial remodeling. We aimed to investigate the potential benefits of allopurinol on atrial remodeling and atrial fibrillation (AF) promotion in alloxan-induced diabetic rabbits.

METHODS 48 alloxan-induced diabetic rabbits were randomly divided into three groups (16 for each): diabetic group (DM group), diabetic allopurinol treatment group (DA group, 25mg/day/kg) and control group (C group). Eight rabbits in each group were respectively used to electrophysiological study, and another eight rabbits from each group were monitored hemodynamics and recorded SBP, DBP and LVEDP and then sacrificed to histological study and western blotting analysis. After 8-week treatment, isolated Langendorff

perfused rabbit hearts were used to evaluate atrial electrophysiological parameters and vulnerability to AF which examined by burst and S1S2 pacing. Collagen volume fraction (CVF) was calculated by Masson staining. Western-blot analysis was applied to assess atrial protein expression of ERK, P38, p-P38, JNK, TGF- β and NF- κ B p50 in left atrial tissue.

RESULTS There were no significant difference regarding SBP, DBP, LVEDP and AERP in the three groups. Intra-atrial conduction time (IACT) was prolonged and AF inducibility was increased in DM group (41/240 vs. 3/240, $P < 0.05$) compared with controls, which were markedly reduced by allopurinol (41/240 vs. 4/240, $P < 0.05$). Allopurinol also attenuated atrial structural remodeling, with significant reduction in CVF. Western-blot analysis revealed DM increased protein expression of ERK, P38, p-P38, JNK, TGF- β and NF- κ B p50, which were reduced by allopurinol.

CONCLUSIONS Allopurinol suppresses AF promotion by preventing atrial electrical and structural remodeling, which may be due to its anti-inflammatory and anti-oxidative characteristics. These results suggest that XO activation may play an important role in the diabetes-related atrial remodeling.

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Study on the Molecular Genetic between Hypertrophic Cardiomyopathy and FKBP12.6

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OBJECTIVES The study aims to explore whether the FKBP12.6 is the causative gene of HCM, further more to provide new sight for HCM in molecular genetics research, clinical diagnosis and treatment.

METHODS In reference to Chinese experts consensus in 2007, American Cardiology Foundation / America Heart Association (ACCF/AHA) in 2011 and European Society of Cardiology (ESC) issued guidelines for the diagnosis and management of hypertrophic cardiomyopathy. The study was approved by hospital ethics committee and all subjects were gave a written informed consent, we enrolled in Thirty seven HCM patients and collected the clinical data and peripheral venous blood (2-5 mL). Genome DNA was extracted from peripheral venous blood. All exons and exon-intron junctions of the FKBP12.6 gene were screened to determine whether there are gene variances via direct sequencing. Control group: two hundred healthy individuals with same ethnic background.

RESULTS

- Thirty seven patients with HCM were enrolled in the study. There are twenty three men and fourteen women, the mean age of these patients was 54.24 ± 15.84 years. Two patients with definite family history of HCM, besides one patient with family history unexplained sudden death. The echocardiogram or cardiac magnetic resonance revealed that thickness of interventricular septal was 19.34 ± 3.903 mm, thickness of left ventricular posterior wall was 11.0 (10.00, 13.00) mm and ten patients with left ventricular outflow tract obstruction. ST-T change was the most common manifestation on ECG, the incidence was up to 72.97%, atrial arrhythmia up to 32.4%, ventricular arrhythmia up to 29.73%, block and Wolf-Parkinson-White syndrome up to 5.41%, pathological Q wave up to 10.81%.
- One patient with two single nucleotide polymorphisms (SNP) was identified by gene sequencing, including FKBP12.6- c.4161T > C (NM_004116.3:c.38-36T > C), T-to-C substitution at nucleotide site 4161, c.11278A > T (NM_054033.2:c.198+ 57A > T), A-to-T substitution at nucleotide site 11278. The above SNP were located at exon-intron junctions. The patient was 35-year-old female was diagnosed as asymmetric hypertrophic obstructive cardiomyopathy, cardiac arrhythmias: atrial Beckoning tachycardia, atrial premature beat. The main symptom was recurrent syncope in 13 years, without previous history of hypertension, diabetes, hyperthyroidism history, The echocardiogram showed that thickness of interventricular septal was 20mm, thickness of left ventricular posterior wall was 9mm and SAM sign was positive.

CONCLUSIONS This study is the first to explore the molecular genetic relationship between FKBP12.6 gene and HCM. We find that two SNP located at the intron of FKBP12.6 gene, which is lower than the frequency of healthy people reported by 1000 Genomes Project Database. In our conclusion, FKBP12.6 gene may not be the causative gene of HCM, but it still needs to increase the sample size to further confirm.