blood were reduced in MI-ablation dogs, but only slight changes in ventricular tissue growth were induced.

Conclusions: The coronary sinus and great cardiac vein peripheral nerve ablation reduce the occurrence of VA and improve ventricular electrical stability, with no obvious effect on heart rate and systemic arterial pressure as well as infarct size. Therefore, local cardiac sympathetic ablation may protect from ventricular arrhythmias during AMI.

GW25-e2199

A Novel Drug-eluting PLLA/ACP Bioabsorbable Scaffold for Coronary Application: Preliminary Experience in Porcine Coronary Arteries

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Objectives: To assess the safety and biocompatibility of a novel bioabsorbable drug-eluting PLLA/ACP scaffold for coronary applications.

Methods: A total of 12 stents, half biodegradable drug-eluting stents (BDS, n=6), half novel fully bioabsorbable drug-eluting scaffolds [NBFs (PowerStent,Absorb), n=6], were randomly implanted into the coronary arteries of 12 pigs. After the operation, histology and drug elution profile in the 28th day, the pigs were sacrificed. Stented segments were processed for quantitative histomorphometry. Histomorphometric analysis was performed to evaluate endometrial hyperplasia and endothelialization. Immunohistochemical assay was applied for expression of NF-κB, β-2-SM-actin, CD31 and eNOS.

Results: At 28-day follow-up, no in-stent restenosis and stent thrombosis were detected in either group. The level of VEGF and NO in BDS group were higher than that BDS group respectively (t(309.86)<0.48, P<0.05). No significant difference among them (F(666.05)<1). No in-stent restenosis was successfully detected in either group. The level of VEGF and NO in BDS group were higher than that BDS group respectively (t(309.86)<0.48, P<0.05). No significant difference among them (F(666.05)<1). No in-stent restenosis was successfully detected in either group.

Conclusions: BDS group and NBFs group are both safe, and BDS group is better.

GW25-e4163

Effects of Astragaloside on Cardiac Function of rats with Cecil Ligation and Puncture and Underlying Mechanism

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Objectives: We aimed to investigate the effect of Astragaloside on cardiac function and to search for possible mechanism in septic rats.

Methods: Fifty male Sprague-Dawley (SD) rats were randomized into Sham-operation group (Sham group, n=10), saline group (NS group, n=20) and Astragaloside group (AST group, n=20). Polymicrobial sepsis model was induced by cecal ligation and puncture (CLP) in NS and AST group. Rats in NS group were injected with saline (10ml/Kg) 2h after CLP and then once a day for 7 days. Rats in AST group were injected AST (25mg/Kg) instead of saline at the same time points. Dead time of rats was recorded every day and cardiac function was tested by ultrasound. Serum concentrations of monocyte chemotractant protein-1 (MCP-1), brain natriuretic peptide (BNP) and cardiac troponin I (TnI) was observed. The left ventricle samples were collected for histomorphology. Myocardial apoptosis on the left ventricle were detected by TUNEL staining in situ, and the changes of Bcl-2 and Bax protein were detected by western blot. Meanwhile, the expression of iNOS, AMPK mRNA and protein were tested.

Results: On day 7 after CLP, 3 out of 20 AST treated rats survived, but all rats in NS group died. Compared with the NS group, AST group could obviously increase ejection fraction (EF) (57.9±5.66>70.6±4.02, P<0.05) but decrease the left ventricular internal dimension in systole (LVIDs) (4.45±0.35>3.44±0.27, P<0.05) and MCP-1, BNP, Ctnrd release in serum (104.8±6.5>89.74±8.2, 2.79±0.21 vs 2.24±0.17, 1.38±0.07 vs 0.91±0.05, P<0.05). Correspondingly, histomorphometry indicated that myocardial inflammation of AST group was comparatively mild. Also, AST treated rats significantly decreased myocardial apoptosis index and the expression of Bax protein was obviously decreased but markedly increased the expression of Bcl-2 protein. Meanwhile, the mRNA expression of iNOS and AMPK in AST treated rats was obviously increased.

Conclusions: AST could effectively extend the survival time, significantly asssuage the cardiac function decrease and markedly myocardial necrosis and apoptosis in septic rats induced by CLP, which may be relative with the expression of iNOS and AMPK.

GW25-e5204

Carboxyl-terminal polypeptide of Cardiotrophin-1 promoted cardiac hypertrophy and induced myocardial remodeling in Kunning mice

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Objectives: To study histiastic characteristics of myocardial remodeling induced by chronic exposure to cardiotrophin-1 in Kunning mice.

Methods: Sixteen amino acids from carboxyl-terminal of mouse CT-1 were selected and synthesized to a polypeptide (Cardiotrophin-1, CT-1-CP), and then were injected intraperitonially to the Kunning mice for 1 to 4 weeks (6 groups; n=10, 5 male), the control group (n=10, 5 male) received intra-peritoneal injection of physiological saline for 4 weeks. At every end point of injection the body weight and height were measured, and the hearts tissues samples were isolated and embedded in paraffin for preparing tissue section. The HE and MASSON staining were performed. The endomterial hyperplasia and endothelialization were evaluated by immunohistochemistry. The level of VEGF and NO in cardiac tissue was detected by western blot. The expression of iNOS and AMPK was detected by TUNEL staining in situ, and the changes of Bcl-2 and Bax protein were detected by western blot.

Results: The increase of body weight at first and second week was higher in the control group than that of the CT-1-CP injected-groups, but was significantly lower than that of the latter at third (t(4.821, P<0.01) and forth week (t=2.019, P=0.05). Also, the body weight of the four-week group had exceeded slightly the control group; the heart weight of mice in CT-1-CP injected-groups were higher than that of the control group, but the heart to body weight ratio has no significant difference among them (F=1.0833, P>0.05). Intraperitonal injection of CT-1-CP one week after injection, the mice began to appear enlarging of ventricular cavity and thinning of the ventricular wall, and the derangement myofibril and the blurred cross striation complicated with uneven staining of cytoplasm, which were scattered in the ventricular wall, were detected. After 2 weeks, the anatomical change of ventricles became hearted and local hypertrphy and ventricular wall expansion, and the focuses of pathological changes of cardiomyocytes increased. 3, 4 weeks later, the lesion was more obvious and the scope of focuses gradually expanded. In focuses with serious pathological change some myocytes were fragmented and some sarcomeres were partially or wholly lost. Masson staining also shows exaggeraded growth of the connective tissues surrounding myofibril and in the spaces where the myofibril array disturbed or sarcernme detected.

Conclusions: Long-term exposure to CT-1-CP could promote cardiac hypertrophy and lead to cardiac remodeling in Kunning mice as well as the ultrastructure damage of cardiomyocyte in remodeled myocardial tissues, otherwise, induced the hyperplasia of fibril-connective tissues.

GW25-e0840

Inhaled Budesonide for the prevention of acute mountain sickness in unacclimatization young men: a double-blind randomized controlled trial

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Objectives: Oral glucocorticoids can prevent Acute Mountain Sickness (AMS); however, these drugs are associated with multiple systemic side effects. The effects of inhaled Budesonide, an alternative AMS therapy, remain unknown.

Methods: The 80 healthy young male plain residents (17~33 years old) were recruited. Potential participants were excluded if they had a high altitude (>2500 m) exposure history in the last year or organic diseases such as congenital heart disease, arrhythmia, liver or kidney dysfunction, psychological or neurological disorder. The subjects were randomly assigned to receive inhalation of budesonide (BUD, 200 µg, bid), procaterol tablet (PT, 25 µg, bid), inhalation of budesonide/locometer (BUD/FFM, 160 µg/4.5 µg, bid) or placebo (1 tablet, bid) (n = 20 subjects, respectively). Subjects began three treatment days before an ascent to 3700 m from 500 m plain within 2.5 h by air. The treatment stopped after arrival. The Lake Louis AMS questionnaire, blood pressure (BP), heart rate (HR), and oxygen saturation (SpO2) were scored at 20 h, 72 h and 120 h following exposure to high altitude. Pulmonary function was measured after 20 h exposure.

Results: Compared with placebo, BUD reduced the incidence of AMS (70% vs. 25% at 20 h after exposure, P<0.05; 10% vs. 5% at 72 h, P>0.05; 10% vs. 5% at 120 h, P>0.05) without side effects, relative risk is 0.357, and the attributable risk is 0.45. SpO2 was higher in BUD, BUD/FFM and PT groups compared to placebo at 20 h (P = 0.0001). All subjects’ SpO2 dropped following ascent (98.1% to 88.12%, P<0.01) and increased gradually but still lower at 120 h than that at plain (92.04% vs. 98.1%, P<0.01). Pulmonary function was not different among the four groups at 20 h. There was no HAPE or HACE reported.

Conclusions: BUD can prevent AMS without side effects. The alleviation of AMS may be related to increased SpO2 rather than pulmonary function.

The registration number of this clinical trial was ChiCTR-PRC-12002748.