CRT-602

Impact Of High Lipoprotein(a) Levels On 5-year Clinical Outcomes Following Percutaneous Coronary Intervention With Drug-eluting Stent In Asian Population

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BACKGROUND It has not been examined whether high Lp(a) levels are able to predict long-term adverse cardiovascular outcomes in patients (pts) undergoing percutaneous coronary intervention (PCI) with drug-eluting stents (DESs). The aim of this study is to assess the impact of Lp(a) levels on the major clinical outcomes following PCI with DESs.

METHODS A total of 674 consecutive pts with angina pectoris who underwent elective PCI with DESs from 2004 to 2009 were enrolled. The pts were divided into two groups according to the levels of Lp(a): Low Lp(a) group [Lp(a)<50 mg/dL, n=559], and High Lp(a) group [Lp(a)≥50 mg/dL, n=115]. The clinical outcomes were compared between the two groups.

RESULTS The incidence of any myocardial infarction (MI), revascularization, target lesion revascularization (TLR), target vessel revascularization (TVR), All-major adverse cardiac events (MACEs), and stent thrombosis was higher in the High Lp(a) group as compared to Low Lp(a) group. However, the incidence of total death and cardiac death were similar between the two groups. In multivariate logistic regression analysis adjusted by gender, age, diabetes mellitus, hypertension, hyperlipidemia, smoking, multivessel disease, LDL-cholesterol, Lp(a)≥50 mg/dL was significantly associated with the 5-year adverse clinical outcomes including any MI, revascularization, TLR, TVR, and All-MACEs.

CONCLUSIONS In our study, angina pectoris pts with high Lp(a) level≥50 mg/dL undergoing elective PCI with DESs in Asian population was significantly associated with adverse clinical outcomes at 5 years.

Table. Five year clinical outcome qccording to Lp(a) groups

	Low Lp(a) (n=559)	High Lp(a) (n=115)	p-value* (unadjusted)	p-value† (adjusted†)	OR (95% CI)
Total Death, n (%)	13 (2.3)	5 (4.3)	0.221	0.072	3.893(0.885-17.121)
Cardiac death	7 (1.3)	2 (1.7)	0.679	0.575	2.099(0.157-28.104)
Any MI	18 (3.2)	12 (10.4)	0.001	0.010	4.094(1.409-11.898)
Revascularization	71 (12.7)	31 (27.0)	<0.001	0.026	2.162 (1.096-4.264)
TLR	33 (5.9)	18 (15.7)	<0.001	0.002	3.651 (1.625-8.203)
TVR	43 (7.7)	22 (19.1)	<0.001	0.001	3.483(1.615-7.509)
All MACE	85 (15.2)	33 (28.7)	0.001	0.045	1.935 (1.016-3.686)
Stent Thrombosis	7 (1.3)	6 (5.2)	0.005	0.023	5.759 (1.278-25.940)

*: χ2 test
†: Adjusted covariates included gender, age, diabetes mellitus, hypertension, hyperlipidemia, smoking, multivessel disease, LDL-cholesterol, Lp(a)≥50 mg/dL.

RESTENOSIS

CRT-603

Effect of Drug-coated Balloon on Porcine Peripheral Arteries: Physiologic Vascular Function, Safety and Efficacy Preclinical Studies

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BACKGROUND Drug coated balloons have been developed to address some of the shortcomings of stents as implantable prostheses in peripheral vascular intervention. The biological profile and efficacy of DCB are strongly related to the formulation of the coating. This study conducted pharmacokinetic, safety and efficacy studies to characterize the paclitaxel (PTX) balloon coating of the Eurocor FREEWAY DCB.

METHODS AND RESULTS Forty-two domestic swine underwent percutaneous FREEWAY overstretch balloon dilation for 1 and 2 minutes of both femoral and iliac (4-5 and 6-8 mm diameter, respectively) arteries. Tissue paclitaxel concentrations and the vascular function were measured at 1h, 1, 3 and 9 days. Neointimal hyperplasia (NH) was quantified by computerized planimetry 5 weeks post-DCB use in a (DCB vs. non-coated control balloon) pre-clinical study. The peripheral artery tissue drug levels were 141.9±37.9 vs. 566.3±179.9, 43.2±13.6 vs. 149.4±71.8, 23.4±8.4 vs. 30.0±16.6 and 3.2±2.4 vs. 4.0±1.1 ng/mg using 1 vs. 2 minutes balloon inflation time at 1h, 1, 3 and 9 days post-DCB use, respectively. The NH was significantly smaller in the arteries dilated with FREEWAY compared with control balloon. DCB led to impairment of endothelium-dependent vasodilation in a tissue PTX dose-dependent manner.

CONCLUSIONS In a pre-clinical porcine model, PTX tissue concentration from the FREEWAY DCB is directly related to time of balloon inflation. DCB reduced the NH in peripheral arteries compared to uncoated balloons. Vascular function impairment was directly related to tissue PTX concentration.

ANGIOMYOGENESIS, CELL THERAPY, GENE THERAPY

CRT-604

Mesenchymal Stem Cells Exposed to Chronic Hypoxia Preferentially Home to Regions of Myocardial Injury Following Intravenous Injection

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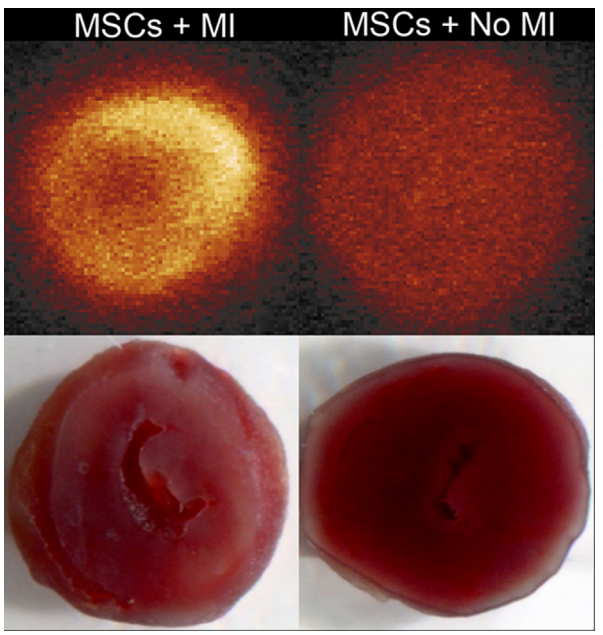
INTRODUCTION Intravenous delivery of MSCs for MI could have major clinical implications. Also, MSCs grown under chronic hypoxic conditions have attributes suggesting more robust healing and homing functions. Therefore, we studied whether human and murine hypoxic MSCs traffic to the heart following myocardial injury and preferentially home to regions of ischemia.

METHODS CD1 male control mice and mice that underwent LAD ligation surgery with 45 minutes of ischemia followed by reperfusion (I/R) received tail-vein injection 24 hours following surgery with either 10e6 indium-111 radiolabeled hypoxic murine or human MSCs grown at 5% O2 (n=10 in each group). Organs were harvested 24 hours post-injection, gamma well counting was performed, and hearts were sectioned and phosphor-imaging was performed to assess signal intensity in the LAD region vs the non-ischemic territory. TTC staining was performed to assess for infarction.

RESULTS For both human and murine MSCs, phosphor-imaging demonstrated preferential homing of radiolabeled MSCs to the LAD region vs. non-ischemic region of the LV following IR while there is no difference in control mice (Figure with phosphor image and corresponding TTC staining below). Following radiolabeled MSC injection, signal intensity was significantly increased in the LAD region vs. posterior wall following IR compared with control mice (34±3% vs 3±1%, p<0.0001). Gamma well counting demonstrated an increase in the number of MSCs to the hearts following IR vs control hearts (3425 ± 264 MSCs/gm vs 1975 ± 160 MSCs/gm, p=0.0002). The number of MSCs per heart significantly correlated with the size of MI by TTC staining (Spearman R 0.61, p=0.004).

CONCLUSIONS Following a murine model of MI, radiolabeled hypoxic human and murine MSCs traffic to the heart and preferentially home to regions of myocardial injury.



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