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Session C5 — *Coronary Surgery*

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The platelet reactivity after percutaneous coronary intervention in patients with double antithrombotic therapy: impact of genetic polymorphisms

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Objectives. To assess antiplatelet therapy efficacy considering the CYP2C19*2 and CYP2C19*3 genetic testing before percutaneous coronary intervention (PCI) in patients with stable coronary artery disease (SCAD).

Method. 55 (SCAD) patients (39 -75 y.o., mean 59±9.68 y.o.) were studied pre- and postoperatively. Beside conventional investigations, antiplatelet therapy efficacy was assessed by Thromboelastography (TEG) and panel Platelet Mapping with arachidonic acid and ADP-induced platelet aggregation and light transmission aggregometry.

Results. Our analysis revealed a normal genotype (GG) in 39 patients, heterozygous polymorphisms CYP2C19 (GA) G681A allele were detected in 14 patients, and 2 patients has homozygous polymorphisms CYP2C19 (AA) G681A allele. All 55 patients in study had normal CYP2C19*3 (Trp212Ter) genotype of CYP2C19 gene. The platelet activation was higher in patients with heterozygous and homozygous carriers comparing to normal genotype. The largest differences were in groups with different genotype (GG, GA, AA), it was maximal amplitude (MA) (p(F) 0,005; p(H) 0,012) and ADP-induced platelet aggregation before PCI (p(F) 0,029; p(H) 0,041). We revealed a statistically significant influence of various genotypes of CYP2C19 * 2 (GG, GA, AA) on the average values of platelet aggregation (p = 0,02). Also a reliable relationship between MACE and the platelet aggregation according to the LTA (p = 0,002) was obtained. The interaction of these two factors was significant (p = 0,022). In our study there was no significant relationship between the different genotypes CYP2C19*2 and the average values of PRU (p=0,057), however, their joint impact on MACE was statistically significant (p = 0,041).

Conclusions. the levels of platelet reactivity higher in patients with heterozygous and homozygous carriers of CYP2C19*2 *versus* normal genotype. Patients with high on-treatment platelet reactivity had a significantly increased risk of having adverse cardiac events.

R&D session

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Synthetic scaffolds with anti-inflammatory effects for vascular prosthesisB. Aliakbarian, E. Arab-Tehrany, A. Hasani, A. Tamayol, D. Palmieri, N. Annabi, A.A. Casazza, P. Perego, A. Khademhosseini, D. Palombo
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Objectives. Cardiovascular diseases cause ~17 million deaths per year globally and this number is predicted to reach 23.3 million by 2030. Tissue engineering holds a great promise to overcome this health barrier by creating functional tissue constructs that can reestablish the structure and function of the injured tissues. However, the fabrication of biodegradable scaffolds that can mimic mechanical properties of the native arterial tissue, support cellular proliferation and activity, and control local inflammation after surgical implant is a major challenge and is the main objective of this study.

Materials & Methods. To address these challenges, we fabricated poly(glycerol-sebacate) (PGS)-polycaprolactone (PCL) electrospun sheets loaded with t-resveratrol to form a vascular prosthesis with anti-inflammatory properties. PCL-PGS electrospun sheets possess mechanical characteristics similar to the native tissue of the cardiovascular system. t-resveratrol is a naturally occurring polyphenolic compound found mainly in grape and red wine and its promising antioxidant and anti-inflammatory effects have been demonstrated previously. We tested two methods for incorporating t-resveratrol into the fabricated electrospun sheets: i) mixing t-resveratrol with the prepolymer followed by electrospinning the mixture and ii) entrapping t-resveratrol within liposomal particles and spraying it on the surface of the scaffold. We measured the physico-chemical and mechanical properties of fabricated scaffolds. We also seeded them with human umbilical vein endothelial cells (HUVEC) and monitored cellular adhesion, metabolic activity, and proliferation over 7 days.

Results & Conclusions. HUVECs cultured on PGS-PCL scaffolds containing nanoliposomes loaded with t-resveratrol showed a higher proliferation in comparison with control samples. Moreover, the expression of pro-inflammatory markers was evaluated. t-resveratrol released from scaffolds down-regulates the basal expression of endothelial pro-inflammatory markers such as Metalloproteinase-9 (MMP-9) and Interleukin-6 (IL-6).

Session C8 — *Coronary Surgery*

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Apelin: a potential marker of coronary artery stenosis and atherosclerotic plaque stability in ACS patientsY Zhou ¹, SB Qiao ¹, Y Wang ²¹Department of Cardiology, Cardiovascular Institute of Fuwai Hospital, Chinese Academy of Medical Sciences and Peking Union Medical College, Beijing, China; ²Department of Cardiology, China-Japan Friendship Hospital, Beijing, China

Objectives. Previous studies have revealed that APJ deficiency can prevent oxidative stress-linked atherosclerosis and that the apelin-APJ system is a mediator of oxidative stress in vascular tissue. Thus, we speculated that the apelin-APJ system is related to arterial atherosclerosis. Recent evidence has shown that the apelin-APJ system is involved in atherosclerosis in mice. Moreover, the involvement of the apelin-APJ system in atherosclerosis in humans has also been investigated. However, the involvement of the apelin-APJ system in acute coronary syndrome (ACS) patients remains unknown. This study was performed to characterize plasma apelin levels following acute coronary syndrome (ACS) and to examine its relationship with coronary stenosis and atherosclerotic plaque stability.

Method. The study enrolled 196 patients admitted with ACS, which was further subdivided into the STEMI group (n=65), non-STEMI group (n=62), and unstable angina (UA) group (n=69), and another 171 outpatients with no coronary heart disease as control. Plasma concentrations of apelin, N-terminal pro-brain natriuretic peptide (NT-pro-BNP) and matrix metalloproteinase-9 (MMP-9) were measured 2h and 6 months after admission, respectively. The coronary angiography characteristics of ACS patients were analyzed by a validated quantitative coronary angiographic system, wherein Gensini scores were obtained, which were computed by assigning a severity score to each coronary segment according to the degree of luminal narrowing and geographic importance. Coronary arteriography was performed immediately after admission of STEMI patients and 3 d to 7 d after admission of UA and non-STEMI patients in the ACS group. The stability and ingredients of atherosclerotic plaque were assessed using an intravascular ultrasound imaging system (IVUS). The ACS group was allocated into two groups, nonruptured and ruptured, according to the plaque characteristics. All statistical analyses were performed using SPSS version 16.0 (SPSS Inc., Chicago, IL, USA). The results were expressed as mean ± SE. Intergroup comparisons were conducted using paired sample *t*-tests for continu-

ous variables and χ^2 test for categorical variables. Multivariate regression analysis was performed to determine the relationship between apelin level and Gensini score as well as between apelin level and plaque characteristics. Statistical significance was considered at $p < 0.05$.

Results. Apelin concentration was reduced compared with healthy controls following ACS (0.54 ± 0.25 vs. 3.22 ± 1.08 ng/mL, $P < 0.001$). The plasma apelin concentrations of the ACS patients increased significantly over time (0.54 ± 0.25 ng/mL at 2 h vs. 0.78 ± 0.36 ng/mL at 6 months, $P = 0.02$) but remained significantly lower than those of the controls (0.78 ± 0.36 ng/mL vs. 3.28 ± 0.94 ng/mL, $P = 0.001$) even at 6 months. The baseline apelin levels were 0.54 ± 0.28 , 0.89 ± 0.13 , and 0.92 ± 0.24 ng/mL in the STEMI, non-STEMI, and UA groups, respectively. The apelin level of the STEMI patients was significantly lower than that of the other two groups ($P = 0.01$, $P < 0.01$). The baseline MMP-9 level (1.07 ± 0.34 ng/mL) of ACS patients was significantly higher ($P = 0.01$) than in the control population (0.54 ± 0.37 ng/mL) and decreased to 0.60 ± 0.53 ng/mL after 6 months, which was not significantly different from the control value. The mean baseline plasma NT-proBNP concentration was 2841.5 ± 152.7 pg/mL, which was significantly higher ($P < 0.001$) than that in the control population (697.2 ± 63.8 pg/mL). Then, the plasma concentration of NT-proBNP decreased to 724.5 ± 54.2 pg/mL at 6 months of ACS, which had no significant difference with the control subjects.

Multiple regression analysis revealed that the Gensini scores of the ACS patients were inversely and significantly correlated with the plasma apelin level ($r = -0.382$, $p = 0.009$) and positively correlated with the plasma MMP-9 level ($r = 0.225$, $p = 0.043$). The NT-proBNP level was not relevant to the Gensini scores.

Moreover, in the ACS patients, apelin levels were significantly lower in the group with the ruptured plaque than in those with the nonruptured plaque (0.42 ± 0.24 vs. 0.68 ± 0.30 ng/mL, $P = 0.042$). Apelin levels were negatively correlated with plaque cross-sectional area (CSA) ($r = -0.425$, $p = 0.018$) and positively correlated with external elastic membrane (EEM) CSA ($r = 0.311$, $p = 0.037$). By contrast, MMP-9 levels were inversely and significantly correlated with lumen CSA ($r = -0.028$, $p = 0.048$) and EEM CSA ($r = -0.346$, $p = 0.021$).

Conclusions. Plasma apelin levels was inversely correlated with the severity of coronary artery stenosis and positively related with the stability of atherosclerotic plaque in humans of ACS. By contrast, MMP-9 levels were inversely and significantly correlated with the stability of atherosclerotic plaque in humans of ACS. Thus, low apelin plasma levels may indicate plaque rupture, which is the major cause of ACS. This study is the first to evaluate circulating plasma apelin levels by comparing control and ACS subjects with regard to the existence of plaque rupture estimated by IVUS observation. These results suggest a cardioprotective function for apelin and a potential therapeutic function for apelin upregulation following ACS.

Session C9 — Aortic Valve

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First experience with proximal scallop on thoracic stentgraft for the treatment of aortic arch aneurysms

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Objectives. Endovascular treatment of thoracic aortic aneurysms (TAA) may be limited by proximal extension to the supra-aortic trunks (SAT). In these cases, SAT might be covered and revascularized by cervical approach or sternotomy. We report our initial experience with the use of a stentgraft with a custom made scallop to allow SAT patency at the proximal aortic neck.

Method. Between September 2012 and November 2013, we treated 10 consecutive patients (5 men, mean age 76 ± 7 years) for TAA involving the arch by a Bolton stentgraft with proximal scallop. Each stentgraft was custom made in an average of 4 weeks, with reinforced scallop detectable by markers to allow alignment with SAT. Median diameter

was 37 mm (34 to 46), and median length was 160 mm (160 to 250); on the scallop, median width was 18 mm (15 to 21) and the median depth 25 mm (13 to 30). The patients were all followed by a postoperative angio-CT, at 6 months and 1 year.

Results. Stents were successfully implanted in all cases without endoleak on intraoperative completion angiography. The scallop was placed next to the ostium of the innominate artery (IA) in one case, IA and left common carotid artery (LCCA) in 6 cases, and LCCA artery and left subclavian artery (LSA) in 3 cases. All targets SAT were patent on completion angiography. LCCA and 1 LSA were revascularized by cervical approach before their coverage, and 2 LSA were occluded by a vascular plug after their coverage. A LSA has been revascularized by a stentgraft through an additional fenestration. Immediate postoperative course was marked by a transient ischemic stroke upon waking, and 1 death from myocardial infarction on day 4. The median hospital stay was 3 days (3-7). No other complication occurred during the median follow-up of 7 months (2-17). All aneurysms appeared excluded without endoleak, with permeable targeted SAT on control angioCT.

Conclusions. Thoracic stentgraft with proximal scallop extends the endovascular treatment options for aneurysms of the thoracic aorta involving SAT. Avoiding associated gestures of revascularization by open surgery, it could represent a significant medical and economic benefit.

Session C17 — Mechanical Circulatory Support

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Invasive treatment of gastrointestinal bleeding in patients with biventricular assist devices – report of two cases

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Background. Gastrointestinal (GI) bleeding is a well-known complication of continuous-flow ventricular assist devices (VAD). Anticoagulation can not be regarded as the only factor of such bleedings. One of the possible explanations is the formation of arterio-venous malformation (AVM) in the intestinal mucosa. The narrow pulse pressure may cause decreased intestinal blood pressure, increased sympathetic tone and dilatation of mucosal veins that can lead to AVM formation. Lower GI AVMs most commonly develop in the caecum and ascending colon. According to the literature, bleedings originated from such AVMs rarely need endovascular or surgical interventions.

Case report. The first patient had extensive anterior myocardial infarction and percutaneous coronary intervention. Due to cardiogenic shock an extracorporeal membrane oxygenator (ECMO) was implanted primarily that was exchanged for biventricular assist device (BiVAD) a few days later with the indication of bridge to transplant. Twenty days later the patient developed serious GI bleeding. After negative gastro-duodenoscopy multiphase CT localized the bleeding in the caecum. Superselective endovascular embolization with polyvinyl alcohol (PVA) particles stopped the bleeding successfully. The second patient had a complicated postoperative course following aorto-coronary bypass operation. On the day of her surgery she underwent coronary angiography for ST elevation, intervention followed by cardiopulmonary arrest and resection. An ECMO was implanted first and some days later a BiVAD with the indication of bridge to recovery. The patient developed massive GI bleeding on the 22nd postoperative day. After excluding gastro-duodenal haemorrhage multiphase CT scan revealed extravasation into the ascending colon. This was followed by superselective endovascular embolization with PVA particles. In four days repeated massive bleeding occurred. Late phases of CT angiogram proved extravasation into the same area of the ascending colon. Superselective embolization with coils was attempted but the bleeding has only stopped temporarily. We performed right hemicolectomy on the next day and the bleeding ceased. Later the BiVAD was removed successfully.