**e0517 FOREARM ARTERIES WITH ULTRASOUND FOR PERCUTANEOUS CORONARY PROCEDURES**

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**Background** The radial artery has become a widely used approach for coronary angiography and intervention in patients, and the ulnar artery is another approach for elective procedure in patients.

**Objective** The objective of this study was to investigate the diameter, peak flow rate of blood flow and anatomy abnormalities of radial arteries and ulnar arteries of left and right forearm and to identify the influencing factors of the diameter of radial arteries and ulnar arteries in Chinese adult patients.

**Methods** 1112 consecutive adult patients who were to accept the selective transradial or transulnar procedures including coronary angiography and percutaneous coronary intervention were enrolled. To examine the radial arteries and ulnar arteries of left and right forearm with colour Doppler ultrasound, to measure the diameter, peak flow rate of blood flow and anatomy abnormalities of radial arteries and ulnar arteries.

**Results** The diameters of right radial artery and ulnar artery were 2.316±0.507 mm vs 2.247±0.518 mm respectively; and the diameters of left radial artery and ulnar artery were 2.324±0.486 mm vs 2.258±0.520 mm respectively, there were no statistical difference (p>0.05). The diameters of radial arteries and ulnar arteries of male patients were larger than those of female patients (p<0.05), but the peak flow rates of blood flow of radial artery and ulnar artery of both sides in male and female patients were similar, and there was statistical difference (p<0.05). The total incidence of anatomy abnormalities of forearm arteries was 26.5%. There were 91.9%, 77.5%, 51.9% and 23.6% of total patients whose right radial artery diameter were larger and equal to the outer diameter of 5 Fr, 6 Fr, 7 Fr and 8 Fr sheathe, respectively. Gender and arm circumference were the influencing factors of diameters of radial arteries and ulnar arteries of forearm.

**Conclusions** The diameters and the peak flow rates of blood flow of radial arteries and ulnar arteries of both sides were similar. The diameters of radial arteries and ulnar arteries in male patients were larger than those in female patients. The incidence of anatomy abnormalities of radial arteries were more than those of ulnar arteries. The gender and arm circumference were the influencing factors of diameters of radial arteries and ulnar arteries of forearm.

**e0519 RAPAMYCIN SUPPRESS KRÜPPEL-LIKE FACTOR 2 EXPRESSION: MECHANISM OF ENDOTHELIAL DYSFUNCTION ASSOCIATED WITH DRUG-ELUTING STENTS**

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**Objects** Although rapamycin released from drug-eluting stents (DESs) affect the antithrombogenic function of endothelial cells, the exactly mechanisms underlying these effects are incompletely understood. We hypothesised that Krüppel-Like Factor 2 (KLF2), a novel and potent regulator of endothelial gene expression, might play an important role in stent thrombosis.

**Methods** We observed the effect of rapamycin on expression of KLF2, endothelial NO synthase (eNOS), tissue-plasminogen activator (t-PA), plasminogen activator inhibitor 1 (PAI-1) and tissue factor (TF) in Human Umbilical Vein Endothelial Cells (HUVECs). Then, with overexpression of KLF2, we mensurated the above mentioned mRNA and protein, respectively. The mRNA and protein were mensurated by RT-PCR and Western Blot Analysis. Furthermore, activation of KLF2 was evaluated by Electrophoretic Mobility Shift Assay (EMSA).

**Results** Rapamycin made the expression and activation of KLF2 strongly reduce by 75.6% and 78.2% so as to induce long-term coronary endothelial dysfunction. In HUVECs, rapamycin made basal eNOS and t-PA decrease by 80% and 87.8%, while making basal PAI-1 and TF increase by 2.5 and 1.5-fold. Overexpression of KLF2 strongly induced eNOS and t-PA expression and reduced PAI-1 and TF expression, reversing protein above-mentioned near to normal state.

**Conclusions** The data indicated that rapamycin strongly inhibited the expression of KLF2, meanwhile, reduced anticoagulants (eNOS and t-PA) and induced procoagulants (PAI-1 and TF). KLF2 played an important role in stent thrombosis owing to rapamycin-induced endothelial dysfunction, which might be a part of mechanism of stent thrombosis associated with DESs.

**e0520 STATIN EFFECTS IN STENT THROMBOSIS INDUCED BY RAPAMYCIN RELEASING FROM DRUG-ELUTING STENTS THROUGH KRÜPPEL-LIKE FACTOR 2 OVEREXPRESSSION**

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**Objects** As we studied before, rapamycin released from drug-eluting stents (DESs) affected the antithrombogenic function of endothelial cells through Krüppel-Like Factor 2 (KLF2) decrease. However, 3-hydroxy-3-methylglutaryl coenzyme A reductase inhibitors (statins) are known to modulate endothelial function by inducing KLF2. Here we report that statin-induced expression of KLF2 can reverse stent thrombosis.

**Methods** We observed the effect of rapamycin on expression of KLF2, endothelial NO synthase (eNOS), tissue-plasminogen activator (t-PA), plasminogen activator inhibitor 1 (PAI-1) and tissue factor (TF) in Human Umbilical Vein Endothelial Cells (HUVECs). Then and KLF2 mRNA was induced by treatment with multiple statins in a concentration-dependent manner. The mRNA and protein were mensurated by RT-PCR and Western Blot Analysis. Furthermore, activation of KLF2 was evaluated by Electrophoretic Mobility Shift Assay (EMSA).

**e0518 A CASE REPORT OF TYPICAL ANEURYSM EXACTLY WITHIN DES IMPLANTED IN LEFT ANTERIOR DESCENDING CORONARY ARTERY**

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This is a case report of left anterior descending coronary artery aneurysm (CAA), typical “zig-zag” phenomenon, that developed exactly within the segment of siroisms drug-eluting stent (DES) implanted after percutaneous coronary intervention (PCI). The patient had a history of rheumatoid arthritis. We speculated the typical coronary artery aneurysms could be related to the vascular inflammatory reaction due to both of his rheumatoid arthritis and DES implantation. For this kind of patients with the history of rheumatoid arthritis, we could get some hint, DES implantation should be careful.
Results  Rapamycin made the expression and activation of KLF2 strongly reduce by 75.6% and 78.2% so as to induce long-term coronary endothelial dysfunction. In HUVECs, rapamycin made basal eNOS and t-PA decrease by 80% and 87.8%, while making basal PAI-1 and TF increase by 2.5 and 1.5-fold. After treatment by statins (especially lovastatin), the expression of KLF2 was increased by 3.8-fold nearly reversing to normal state.

Conclusions  Taken together, these observations indicate that statin-dependent induction of KLF2 provides a new treatment for stent thrombosis induced by rapamycin releasing from drug-eluting stents.

**e0521 CLINICAL EVALUATION OF STATIN THERAPY IN DiABETIC PATIENTS UNDERGOING PERCUTANEOUS CORONARY INTERVENTION**

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Objective  The long-term effect of statin therapy in diabetic patients after percutaneous coronary intervention (PCI) is not well established. Accordingly, we sought to determine whether statin therapy initiated at the time of percutaneous coronary intervention reduces total and cardiac mortality among diabetic patients.

Methods  We collected data from 569 consecutive patients who underwent PCI. We then compared all-cause and cardiac mortality rates in 249 patients with diabetes mellitus of whom 74 (29.7%) were treated with statin at the time of PCI. To adjust the variables that would have been related to the decision regarding statin administration, multivariate Cox regression was carried out.

Results  During follow-up (4.4 ± 1.3 years), 23 patients died (including 12 who died of cardiac causes). The Multivariate analysis showed statin therapy to be significantly associated with reduced cardiac mortality (HR 0.39, 0.16–0.95; p = 0.039), but not with all-cause mortality.

Conclusion  Statin therapy was associated with a significantly reduced risk of cardiac mortality in patients with diabetes mellitus and coronary artery disease after PCI.

**e0522 DUAL ANTIPLATELET PLUS TIROFIBAN THERAPY HAVE A BENEFICIAL EFFECT ON ACUTE CORONARY SYNDROME IN DiABETIC PATIENTS UNDERGOING PERCUTANEOUS CORONARY INTERVENTION**

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Background  Diabetes is strongly associated with clopidogrel resistance, thrombosis and the development of coronary artery disease (CAD). Some trials suggest that inhibition of glycoprotein IIb/IIIa can improve the outcome of clopidogrel resistance in patients undergoing percutaneous coronary interventions (PCIs). However, the efficacy of small-molecule IIb/IIIa receptor inhibitors in acute coronary syndrome (ACS) patients with diabetes undergoing PCI has not been specifically investigated.

Methods  We randomised consecutive ACS patients with diabetes undergoing PCI, to tirofiban or placebo groups along with double antiplatelet therapy. High-dose bolus (20 mg/kg per 3 min) of tirofiban was administered immediately before PCI followed by 8 h continuous infusion (0.15 mg/kg per min). Postprocedural myocardial necrosis was assessed prospectively by measurement of cardiac troponin I (cTnI) at 6 and 24 h after PCI. The primary end-points were post-PCI coronary flow estimated by corrected TIMI frame count and post-PCI myocardial infarction.

Result  138 patients entered the study (66 randomised to placebo and 72 randomised to tirofiban). Post-PCI corrected TIMI frame count was 9.2 ± 3.6 in tirofiban and 13.0 ± 7.6 in placebo groups (p = 0.05). The prevalence of post-PCI myocardial infarction was similar in the two groups (17 vs 26%, p = 0.167, respectively).

Conclusion  Upstream use of tirofiban in ACS patients with diabetes undergoing PCI, along with double antiplatelet therapy, was associated with a decreased risk of distal embolisation.

**e0523 RISK FACTORS OF ACUTE RADIAL ARTERY OCCLUSION FOLLOWING TRANSRADIAL PERCUTANEOUS CORONARY INTERVENTION IN SENILE PATIENTS**

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Background  Compare with transfemoral percutaneous coronary intervention, the access site complication is less with transradial percutaneous coronary intervention (TRI). The acute radial artery occlusion (Acute RAO) is the most common complication following TRI. Although the incidence of acute RAO is less (0–10%) and acute RAO cannot induce upper-extremity ischaemia with positive Allen test tests, it makes re-TRI procedures impossible. To elucidate the risk factors of acute RAO is the best way to prevent it occurring.

Methods  A total of 1256 positive Allen test patients (>60 years old) who underwent TRI (during May, 2004 to May, 2009) were divided into two groups: normal group and RAO group, according to whether the patient without or with acute RAO. Risk factors of acute RAO were analysed by logistic regression model.

Results  Acute RAO occurred in 28 patients (2.2%). Univariate analysis showed, the smaller size of sheath used, the higher incidence of acute RAO occurred. As compared to the patients in normal group, there are more female and diabetes mellitus patients in RAO group. The dose of heparin used in the operational procedure in RAO group were significantly less than normal group (5262 ± 523 IU vs 7425 ± 980 IU, p < 0.01). The post-procedure duration of high-pressure compression haemostasis were longer in RAO group than normal group (354.9 ± 35.4 min vs 264.7 ± 43.2 min, p = 0.03). Logistic regression analyses showed that the dosage of heparin used in the procedure (RR: 2.812, 95% CI 1.161 to 6.782, p = 0.016), the size of sheath (risk ratio: 4.978, 95% CI 3.211 to 10.675, p = 0.001) and the post-procedure compression time (RR: 2.431, 95% CI 1.589 to 3.801, p = 0.004) were independent risk factors for acute RAO.

Conclusion  The incidence of acute RAO can be minimised by proper sheath selection, appropriate anticoagulation used during operational procedure, and avoiding prolonged duration of high-pressure compression haemostasis following the procedure.

**e0524 INTENSIVE CHOLESTEROL LOWERING WITH SIMVASTATIN IMPROVES THE OUTCOMES OF PERCUTANEOUS CORONARY INTERVENTION IN PATIENTS WITH ACUTE CORONARY SYNDROME**

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Objective  To evaluate the immediate protective effects of intensive statin pretreatment on myocardial perfusion and myocardial ischaemic injury during PCI.