Table: Risk factors, biomarkers and echocardiographic parameters according to CACS

Variable	CACS=0 (n=428)	CACS > 0 (n=802)	P value	OR(95% CI)*	P* value
Baseline variables					
Age, years	60.9 ± 13.8	71.2 ± 10.6	< 0.001		
Gender, (male)	248 (57.9)	430 (53.6)	0.146		
Diabetes	117 (27.3)	308 (38.4)	< 0.001		
Hypertension	223 (54.6)	529 (66.0)	< 0.001		
Smoking	76 (17.7)	171 (21.3)	0.206		
eGFR	113.6 ± 44.1	98.5 ± 38.3	< 0.001		
Laboratory biomarkers					
Hemoglobin, g/dL	12.9 ± 1.9	12.2 ± 1.9	< 0.001	0.936 (0.857-1.021)	0.136
Mean platelet volume	10.0 ± 0.8	10.1 ± 0.9	0.606	1.148 (0.971-1.356)	0.105
Glucose	122.9 ± 39.0	129.2 ± 52.1	0.017	0.653 (0.997-1.004)	1.001
HbA1c	6.8 ± 1.5	6.9 ± 3.0	0.140	1.090 (0.938-1.267)	0.261
Uric acid	4.6 ± 1.6	4.9 ± 1.7	0.033	1.064 (1.961-1.178)	0.233
Calcium	8.8 ± 0.7	8.7 ± 0.6	0.001	0.869 (0.698-1.082)	0.210
Phosphate	3.6 ± 0.8	3.4 ± 0.8	0.021	1.043 (0.877-1.247)	0.635
hs-CRP >2.0mg/dL	110 (25.7)	298 (37.2)	< 0.001	1.352 (0.956-1.910)	0.088
Triglyceride, mg/dL	150.9 ± 98.1	148.8 ± 108.6	0.743	1.001 (1.000-1.003)	0.077
Total cholesterol, mg/dL	177.6 ± 41.6	175.1 ± 47.1	0.334	1.002 (0.999-1.006)	0.231
LDL-cholesterol, mg/dL	94.7 ± 30.3	93.1 ± 27.9	0.569	0.997 (0.989-1.006)	0.523
HDL-cholesterol, mg/dL	44.5 ± 13.0	41.1 ± 13.4	0.001	0.984 (0.970-0.997)	0.018
Echocardiographic parameters					0.000
LV mass	154.9 ± 37.0	168.2 ± 48.2	< 0.001	1.011 (1.007-1.016)	<0.001
E/E'	11.6 ± 3.6	13.4 ± 4.5	< 0.001	1.075 (1.026-1.126)	0.002

* multivariate logistic regression adjusted by gender, age, diabetes, hypertension, smoking, eGFR

TCTAP A-198

Elevated Plasma HsCRP Level Is a Predictor of Periprocedural Myocardial Injury During PCI

Mohsin Ahmed, Nazir Ahammed Chowdhury, Afzalur Rahman, Mostafizur Rahman
National Institute of Cardiovascular Disease, Dhaka, Bangladesh

Background: Elevated high sensitivity C-reactive protein (hs-CRP) has been well known as a biomarker reflecting inflammatory process for prediction of ischemic events among patients with coronary artery disease. Relatively small studies have identified a heightened and sustained inflammatory response after PCI to be a predictor of periprocedural events after coronary angioplasty and a marker of increased restenosis risk among patients undergoing coronary stenting. Embolization of atherosclerotic and thrombotic debris can induce myocardial necrosis during percutaneous coronary interventions (PCIs). This study was designed to evaluate whether pre procedural hs-CRP level is associated with procedure related distal microembolization producing myocardial injury (assessed by CK-MB level) after percutaneous coronary intervention (PCI).
Methods: A total of 310 patients with chronic stable angina and acute coronary syndrome, who would undergo elective percutaneous coronary intervention were evaluated in National Institute of Cardiovascular Disease (NICVD), Dhaka with a view to evaluate the relationship between preprocedural hs-CRP and rise of CK-MB level, before and after PCI. Patients were divided in 2 groups according to hs-CRP: Group I = hs-CRP < 3 mg/ L, Group II = hs-CRP > 3 mg/ L.
Results: A total of 310 who were divided into two groups: Normal CRP group (n = 131) and elevated CRP group (n= 179). Following PCI, CK-MB level was raised from baseline in both groups. In normal CRP group, there was no significant elevation of CK-MB level after PCI (Pre-procedural VS Post-procedural: 18.6 ± 5.4 VS 29.1 ± 5.6 mg/L, p= ns). In elevated CRP group, there was significant elevation of CK-MB level after PCI (Pre-procedural VS Post-procedural: 19.1 ± 6.7 VS 52.46 ± 9.4 mg/L, p < 0.01). The mean rise of CK-MB level was higher in group II than group I (33.06±11.62U/L vs. 11.52±9.60 U/L). The findings were statistically significant between the study groups (p>0.05). Also there was a positive linear correlation between preprocediral hsCRP level and rise of CKMB (r=0.22) following procedure and it was statistically significant (p<0.05). Multivariate logistic regression analysis done among traditional predictors of PCI outcome advanced age (>50 years), female gender, diabetes mellitus, BMI, hypertension, smoking, dylipidaemia, Type C lesion, multiple stents, post dilatation and hsCRP. After removing the effects of the all other variables, hs- CRP was independent predictors of periprocedural myocardial injury during PCI, assessed by CK-MB elevation with OR 1.57 and p=0.001.
Conclusion: The inflammatory activity, as represented by hs-CRP level, is associated with procedure related microvascular injury as assessed by CK-MB elevation after PCI. Measurement of hs-CRP levels could provide a rationale for risk stratification before coronary intervention and may be a useful tool to target aggressive anti-aggregatory or anti-inflammatory therapy to patients that are exposed to the highest risk for ischemic complications. As distal microembolization is a determinant of short and long term mortality after PCI, specific strategies may be developed to minimize myocardial injury in subjects with elevated hs-CRP level.

TCTAP A-199

Interaction Between Renin-angiotensin-aldosterone System Genes and Serum Uric Acid on Blood Pressure

Chen Xin
Shanghai Sixth People's hospital, Shanghai, China

Background: Hemodynamic changes in central aorta were independently associated with target organ damage and cardiovascular diseases. Central blood pressure (BP) may be more predictive than brachial BP. Understanding the interaction between genetic and environmental factors contributing to BP is an important issue in view of the relation of hypertension with outcome. Genetic variation of renin-angiotensin-aldosterone system (RAAS) genes is closely related to the susceptibility in hypertension, and plays an important role in the changing of artery structure and function in hypertensives. Moreover, because the pathophysiological role of central aortic and brachial BP is different, genetic variations maybe have different effects on the regulation of central and brachial BP. Using the standardized genetic epidemiological methods in the general population, we studied the association of two single nucleotide polymorphisms (SNPs) in RAAS genes including angiotensin II type 2 receptor (AT2R, G1675A polymorphism), angiotensinogen (AGT, C-532T polymorphism) with central and brachial BP and hypertension. We also studied the interaction between genes and environmental factors, in relation to central BP, brachial BP and hypertension.
Methods: In 1293 residents of Zhejiang province, we conducted a questionnaire survey, collected blood, estimated central blood pressure using the SphygmoCor device and genotyped two SNPs in candidate RAAS genes with polymerase chain reaction-restriction fragment length polymorphism (PCR-RFLP) method and Taqman probe method. SAS 9.1 software was used for statistical analysis.
Results: The 1293 participants included 308 (23.8%) hypertensive patients, of whom 106 (34.4%) were taking antihypertensive medication. For AGT C-532T polymorphism, CT and TT compared with CC had lower central pulse pressure (PP) (P=0.048) in all subjects and lower central SBP (P=0.03) and PP (P=0.02) in men. Each T allele was associated with a lower central PP by 2.5mmHg. We found a significant interaction between AGT C-532T polymorphism and AT2R G1675A polymorphism in relation to central SBP (Pint=0.05). With the increase of the AGT C-532T T allele, the A allele carriers of the AT2R G1675A polymorphism had a lower central SBP (P=0.05). We also found a significant interaction between the AGT C-532T polymorphism and serum uric acid in relation to central BP (Pint≤0.03). Compared with AGT C-532T CC subjects central BP was significantly lower in T allele carriers with a concentration of serum uric acid between 264μmol/l and 319μmol/l (P≤0.04).
Conclusion: First, both in single gene analysis and analyses involving gene-gene, gene-environment interactions, the T allele carriers of AGT C-532T polymorphism (except women) showed a lower central SBP and PP. AGT C-532T polymorphism may be associated with hypertension and arterial stiffness. Second, the interaction between AGT C-532T polymorphism and AT2R G1675A polymorphism influenced central SBP. With the increase of the AGT C-532T T allele, the A allele carriers of the AT2R G1675A polymorphism had a lower central SBP compared with GG homozygotes. The AT2R G1675A polymorphism may be involved in the blood pressure regulation. Genetic variants of RAAS might interact with environmental factors in the regulation of central BP, pathogenesis of hypertension and arterial stiffness.

TCTAP A-200

The Diffuse and Multi-vessel Coronary Artery Spasm Increase the Incidence of Recurrent Chest Pain During 3-year Clinical Follow up

Sang-Ho Park[1], Seung-Woon Rha[2], Won-Yong Shin[1], Dong-Kyu Jin[1],
Byoung Geol Choi[2], Se Yeon Choi[2], Yoonjee Park[2],
Akkala Raghavender Goud Hu Li[2], Sunki Lee[2], Ji Bak Kim[2], Sung Il Im[2], Jin Oh Na[2],
Cheol Ung Choi[2], Hong Euy Lim Lim[2], Jin Won Kim[2], Eung Ju Kim[2],
Chang Gyu Park[2], Hong Seog Seo[2], Dong Joo Oh[2]
[1]Soonchunhyang University Cheonan Hospital, Cheonan, Korea (Republic of),
[2]Korea University Guro Hospital, Seoul, Korea (Republic of)

Background: There are limited data how whether there are differences in clinical outcomes according to spasm extent (focal versus diffuse) and spasm type (single versus multi-vessel).
Methods: A total 2812 patients (pts) underwent coronary angiography with acetylcholine (Ach) provocation test from Nov 2004 to Oct 2010 from prospective spasm registry were enrolled. The pts were categorized into three groups; negative spasm test group (n=1178), other positive group (n=1098), diffuse and multivessel spasm group (n=536). The definition of diffuse spasm was defined as longer than 30mm in length of significant spasm area. Other positive spasm group consisted of total positive Ach provocation test groups except diffuse and multi-vessel spasm group. Cumulative clinical outcomes up to 3 years were compared among the 3 groups.
Results: In proportional hazard Cox-regression analysis adjusted by co-variates such as age, gender, current smoking, current alcoholics, myocardial bridge, HDL cholesterol, hypertension, diabetes, and dyslipidemia, diffuse and multivessel spasm group had the higher incidence of recurrent chest pain requiring repeat coronary angiography during 3-year follow-up, compared to other positive spasm group (HR, 0.634; 95% CI, 0.431-0.931; p-value, 0.020) and negative spasm group (HR, 0.505; 95% CI, 0.336-0.758; p-value, <0.001). However, there was no difference between other positive spasm group and negative spasm group (Figure).
Conclusion: The pts with diffuse and multivessel spasm was associated with higher incidence of adverse clinical outcomes such as recurrent chest during 3-year follow-up period.

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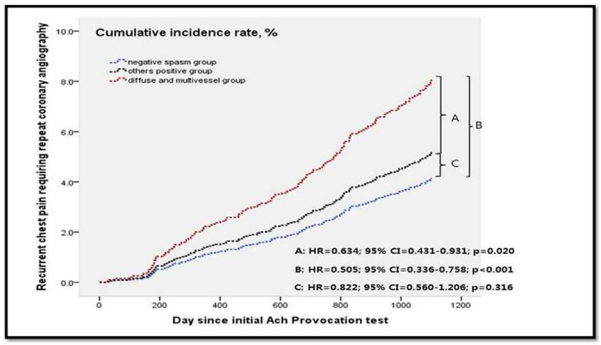

Figure. Proportional hazard Cox-regression analysis adjusted by co-variates, diffuse and multivessel positive spasm group had the higher incidence of recurrent chest pain requiring repeat coronary angiography during 3-year follow-up period, compared to other positive spasm group (HR, 0.634; 95% CI, 0.431-0.931; p-value, 0.020) and negative spasm group (HR, 0.505; 95% CI, 0.336-0.758; p-value, <0.001). However, there was no statistic significance between other positive spasm test group and negative spasm test group.

TCTAP A-201

Impact of Simvastatin on Development of New-onset Diabetes Mellitus in Asian Population: Three-year Clinical Follow up Results

Yoonjee Park, Seung-Woon Rha, Byoung Geol Choi, Se Yeon Choi, Cheol Ung Choi, Hong Euy Lim, Jin Won Kim, Eung Ju Kim, Chang Gyu Park, Hong Seog Seo, Dong Joo Oh
Korea University Guro Hospital, Seoul, Korea (Republic of)

Background: Although statin therapy is beneficial for vascular diseases, the relationship between specific statin therapy and incidence of new-onset diabetes mellitus (DM)remains uncertain. We evaluated the impact of Simvastatin therapyon the development of new-onset DM from3-year clinicalfollow up datain a series of Asian population.
Methods: A total of 3,436 consecutive patients who did not have DM were enrolled. New-onset DMwas defined as having a fasting blood glucose ≥126mg/dLor HbA1c ≥6.5%. Baseline characteristics between the Simvastatin and the control group were propensity score matched (PSM, C-statics=0.808). Three-year cumulative incidence of new-onset DM was compared between the two groups.
Results: At baseline, patients in the Simvastatin group showed higher prevalence ofelderly, male gender,dyslipidemia, coronary artery disease, smoking and alcoholic history, and higher levels of HbA1c, fasting glucose, triglyceride, fibrinogen, and ALP. Three-year clinical follow up results showed a higher incidenceof new-onset DM in the Simvastatin group (3.8% vs. 2%, p=0.017). Following PSM (C-statics=0.808), the 2 groups were well balanced except for higher levels of fibrinogen, ALT, and ALP in the Simvastatin group. After adjustment, there was no difference in the incidence of new-onset DMbetween the 2 groups up to 3years (Figure).
Conclusion: In our study, the relationship between the use of Simvastatin and the incidence of new-onset DM remains unclear. Long-term follow up with a larger study population will be necessary for further information.

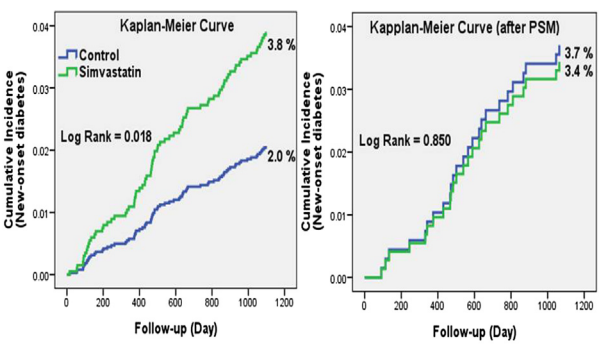

Figure. Impact of Simvastatin on new-onset DM

TCTAP A-202

Is Acetyltransferase Activity an Indicator of BMS Condition in the Follow-up of Coronary Stenting

David G. Iosseliani, Irina E. Kuznetsova
City Center of Interv. Cardioangiology, Moscow, Russian Federation

Background: In-stent stenosis remains one of the most thrilling problems of interventional cardiology, and the search of the ways of its solution includes the study of causes and mechanisms of neointimal hyperproliferation. For this reason the study of the impact of N-acetylation phenotype on the process of restenosis seems very interesting.
Methods: Retrospective study included 100 male patients aged 56,8 ± 6,1 years on the average, who received 116 coronary BMS for chronic CHD from December 2003 to January 2007. The patients have been selected for the study after control coronary angiography performed in 7,2 ± 2,2 months after PCI. The main inclusion criterion was the presence of in-stent stenosis (Group 1, n=50) and good mid-term results (Group 2, control, n=50) in the absence of known clinical and angiographic risk factors for restenosis development. Baseline angiographic data of patients and immediate results of PCI were evaluated by two independent experts. The standard Sulphadimine was used as the test-agent. After single peroral intake of 500 mg of Sulphadimine, the urine has been collected for 6 hours, and than the ratio of pro-metabolized (N-acetyl-sulphadimine) and non-metabolized sulphadimine in urine was determined with the help of high-effective liquid chromatography.
Results: Among the studied patients, there were 38% slow acetylators and 62% fast acetylators. The analysis of the distribution of acetylation phenotype on groups 1 and 2 revealed high statistically significant prevalence of fast acetylators among patients with in-stent stenosis in the mid-term after PCI, P=0,0006.
Conclusion: We revealed reliable direct correlation between the velocity of acetylation processes and the degree of in-stent stenosis after coronary stenting with BMS in patients with chronic coronary heart disease.

TCTAP A-203

The "4S" Law for the Management of Iatrogenic Aortocoronary Dissection: Clinical Results and Importance of Immediate Bail-out Stenting

Shao-Ping Nie, Edmundo Patricio Lopes Lao, Xiao Wang, Yan Qiao, Chang-Sheng Ma
Beijing Anzhen Hospital, Capital Medical University, Beijing, China

Background: Iatrogenic aortocoronary dissection is a rare but potentially disastrous complication of percutaneous coronary intervention (PCI). However, there is a paucity of data on the optimal initial management of this complication.The aim of this study was to depict the characteristics and causes of iatrogenic aortocoronary dissection and to elucidate the importance of a new strategy based on immediate bail-out stenting.
Methods: We collected 18 cases of iatrogenic aortocoronary dissection during PCI in 8 cardiac catheterization centers between 2005 and 2013, which were managed by a single senior operator. A "4S" law based on immediate bail-out stenting performed within 15 min was the initial strategy in most cases. The characteristics, treatment, and in-hospital outcomes of the patients were evaluated.
Results: According to the NHLBI classification, 15 patients (83.3%) had extensive dissection (Type D to F) with acute closure (Type F) in 4 patients. In 3 patients (16.7%), the dissections were diagnosed during coronary angiography, and in 15 patients (83.3%), during PCI. From the 18 patients, 16 patients (88.9%) underwent bail-out stent implantation, with technical success in 14 patients. Among them, 13 patients followed the "4S" law, with 7.7 ± 3.2 min from onset of dissection to completion of bail-out stenting, and the procedural success rate was 100% with no deaths. Two patients died due to time delay (stenting > 15min) and the third one died due to hemodynamic collapse because the guidewire failed to cross the true lumen.
Conclusion: This study indicated that the "4S" law based on immediate bail-out stenting is a feasible and efficient strategy for the management of iatrogenic aorto-coronary dissection.

TCTAP A-204

Abdominal Adiposity Measured with Dual Bioelectrical Impedance Analysis of Hospitalized Patients in the Cardiology Ward

Kenji Sadamatsu, Kaoru Takegami, Taku Matsuura, Arihide Okahara, Takaharu Shirahama, Daigo Mine, Yasuaki Koga, Yasutsugu Nagamoto, Keiki Yoshida
Saga-ken Medical Centre Koseikan, Saga, Japan

Background: Obesity is a well-known risk factor of cardiovascular disease. However, several reports demonstrated "obesity paradox", while central obesity was associated with the highest risk of mortality, which suggests the significance of visceral fat measurement. Recently, a novel system with dual bioelectrical impedance analysis (HDS-2000, Omron healthcare, Kyoto) was developed. This technique does not need radiation exposure and the measurements were validated to have a good correlation with computed tomography.
Methods: A total of consecutive 60 patients (69.3 ± 10.6 years old, male 80%) admitted to the department of cardiology were measured their visceral fat areas with dual bioelectrical impedance analysis. Control group included 23 healthy volunteers (31.5 ± 8.3 years old, male 57%).
Results: The values of body mass index (24.0 ± 4.6 vs 21.6 ± 2.6, p=0.03) and visceral fat area (82.0 ± 51.6 vs 46.1 ± 15.7 cm^2, p<0.01) in the hospitalized patients

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