in the fibrinolysis group, relative risk of 0.40 [95% confidence interval, 0.16-1.01], P=0.053. Results after 1 year will also be presented.
Conclusions: The results of STOPAMI-2 trial show that in patients with acute myocardial infarction, stenting plus abciximab increases the chances of a favorable clinical outcome as compared to fibrinolysis plus abciximab. The significantly greater degree of myocardial salvage is the mechanism of the clinical benefit yielded by stenting plus abciximab.

POSTER SESSION

1076 Stable Ischemic Syndrome III: Pathogenesis

Monday, March 18, 2002, 9:00 a.m.-11:00 a.m.
Georgia World Congress Center, Hall G
Presentation Hour: 10:00 a.m.-11:00 a.m.

1076-38 Upregulation of Angiotensin Subtype Receptor 1 (AT1) and Beta Adrenergic Receptor (Beta 1-AR) and Downregulation of AT2, Transforming Growth Factor Beta 1 (TGF-Beta 1), and TGF-Beta Receptor II in Intimal Hyperplasia of Intramyocardial Coronary Arteries Distal to a Severe Epicardial Coronary Stenosis

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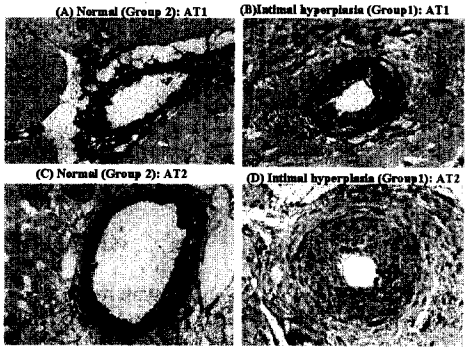
We have described inward remodeling with intimal hyperplasia and luminal narrowing of small intramyocardial coronary arteries (SIMCA) distal to a severe epicardial coronary stenosis in pigs. To examine whether changes in AT1, AT2, β1AR, β2AR, TGF-β1 and TGF β- R II are involved in this arterial remodeling process, we studied 10 pigs with a 4-week severe LAD stenosis that reduced flow by ~30% (Group 1), and 8 pigs without coronary stenosis (Group 2). The outer area and lumen area of SIMCA were measured; the ratio of lumen/outer area was defined as the % lumen area and used as an index for the degree of lumen narrowing. Immunochemical staining was employed to evaluate vascular smooth muscle cell (VSMC) proliferation (ki-67) and expression of AT1, AT2, β1-AR, β2-AR, TGF-β1 and TGF-β R II in SIMCA walls. **Results:** The % lumen of SIMCA in Group 1 deceased compared with Group 2 (17±15% vs 38±13%, p<0.05). Ki67-positive cells in SIMCA of Group 1 increased (5.8 ± 0.5% vs 0.9 ± 0.3%, P<0.01). Expression of AT1 and β1-AR in VSMC of SIMCA increased (Figure A & B), whereas AT2 (Figure C & D), TGF-β1 and TGF-β R II decreased in Group 1. **Conclusions:** The growth-promoting factors AT1 and β1-AR were up-regulated and the antiproliferative factors AT2, TGF-β1 and TGFβ R II were down-regulated in intramyocardial coronary arteries distal to a severe epicardial coronary stenosis. This may contribute to the VSMC proliferaion and inward remodeling in the intramyocardial coronary arteries and create further flow restrictions in ischemic myocardium.



1076-39 Impaired Coronary Collateral Vessel Recruitment in Diabetes Mellitus: Evidence for Defective Ischemic Tolerance

Yilmaz Nisanci, Murat Sezer, Berrin Umman, Sabahattin Umman, Huseyin Oflaz, Ercument Yilmaz, Faruk Erzengin, Onal Ozsaruhan, *Istanbul University, Istanbul Faculty of Medicine, Istanbul, Turkey.*

Collateral channel opening is the one of the components of the ischemic tolerance developing during subsequent coronary balloon occlusions. The effect of diabetes mellitus (DM) on coronary collateral recruitment (CR) is still not known We therefore sought the effect of DM on CR in patients with stable angina (SAP) pectoris by using intracoronary pressure measurement technique.
Methods. Study materiel consisted of 44 patients (21 diabetic) with SAP. All of the patients had single vessel disease with more than 70% narrowing and underwent stent implantation to this vessel. Total and subtotal occlusions were excluded. After angiography, fiber-optic pressure monitoring guide-wire was advanced to the stenosis to be dilated. Myocardial fractional flow reserve (FFRmyo) was determined under adenosine hyperemia by the ratio of simultaneously measured mean distal pressure to mean aortic pressure. During subsequent two 1-min. balloon occlusion, distal pressure was recorded as coronary wedge pressure (CWP). Collateral flow index (CFI) was determined by the ratio of simultaneously measured CWP (mmHg) to mean aortic pressure (Pa, mmHg). Percentage of the improvement in the CFI (first to second occlusion) between two occlusions was determined for each patient.
Results
There was no difference between two groups in terms of pre-intervention FFRmyo (0.54 +/- 10.2 in DM group and 0.50 +/- 11.2 in non-DM group). The baseline CFI was significantly higher in non-DM group (0.26 +/- 0.09 versus 0.17 +/- 0.08, p<0.03). Beyond this finding, mean CFI increased by 17 % (from 0.17 +/- 0.08 to 0.20 +/- 0.09) in DM group and by 30 % (from 0.26 +/- 0.09 to 0.34 +/- 0.10) in non-DM group. There was statistically significant difference between these two group in terms of improvement in CFI during subsequent balloon occlusions (p<0.01).
Conclusion
In addition to poor collateral vessels seen in patients with DM, CR is also impaired. This finding suggests that DM abolishes ischemic tolerance in terms of CR as well.

1076-40 Frequency and Determinants of Silent Plaque Disruption in Patients With Stable Coronary Syndrome

Yaqing Sun, Shumei Ma, Kyoichi Mizuno, Masamichi Takano, Kouji Seimiya, Kentaro Okamatsu, Hiroyuki Kamon, Ryota Uemura, Fumiyuki Ishibashi, Shinya Yokoyama, Takayoshi Ohba, *Nippon Medical School, Chiba Hokusoh Hospital, Chiba, Japan, China Medical University, The Second Clinical College, Shenyang, People's Republic of China.*

Background: Although pathologic and clinical studies have suggested that plaque disruption with or without thrombus plays a key role in the development of acute coronary syndrome and sudden progression of atherosclerosis, the prevalence of silent plaque disruption and its correlation with coronary risk factors in patients with stable coronary syndrome are unclear. The study was designed to clarify the two questions using coronary angioscopy.
Methods: Seventy-eight consecutive patients with stable coronary syndrome without any complaints within three months before the angioscopic study were enrolled in this study. We evaluated 49 non-ischemi-related coronary arteries without significant stenosis on arteriography in 41 patients. And the presence of silent plaque disruption and coronary risk factors were recorded.
Results: The frequency of silent plaque disruption was 29.3%. Compared with patients without silent plaque disruption, glycohemoglobinA1c elevated in patients with silent plaque disruption (6.8±0.5% versus 5.4±0.7%, P=0.0003), as was the frequency of diabetes mellitus and hypertension (66.7% versus 10.3%, P=0.001; 83.3% versus 31.0%, P=0.005; respectively). Multivariate analysis revealed that the independent predictors of silent plaque disruption were: diabetes mellitus and hypertension.
Conclusions: In patients with stable coronary syndrome, silent plaque disruption of non-ischemia-related coronary arteries were commonly observed. Its determinants were diabetes mellitus and hypertension. Therefore, control of diabetes mellitus and hypertension may prevent the incidence of silent plaque disruption and reduce the risk of acute coronary syndromes.

1076-41 Homocysteine and Coagulation Factors as a Risk Factor of Myocardial Infarction at a Young Age

Masahiko Saigo, Satoshi Abe, Masakazu Ogawa, Sadatoshi Biro, Shinichi Minagoe, Tsuminori Yamashita, Tatsuru Matsuoka, Hiroyuki Torii, Hitoshi Toda, Yoshihiko Atsuchi, Koushi Mawatari, Ikuro Maruyama, Chuwa Tei, *Kagoshima University, Kagoshima, Japan, Kagoshima CCU Network, Kagoshima, Japan.*

Background: An increased homocysteine (Hcy) has been reported to be one of the risk factors for coronary heart disease. Many investigators recognize the hypercoagulable state in hyperhomocyteinemia, but the mechanisms remain still unclear. Purpose and Methods: To evaluate the importance of Hcy to the pathogenesis of young myocardial infarction (MI), we conducted a case-control study in 97 males with first MI <45yr and 148 age-matched male controls. We measured plasma Hcy and coagulation factors, tissue factor (TF), tissue factor pathway inhibitor, AT-III, protein C, protein S, factor VII, activated factor VII, prothrombin fragment 1+2, and lipoprotein (a). All parameters were divided into quantiles, and conventional risk factors, hypertension, hyperlipidemia, diabetes, and smoking were difined to have or not for statistical analysis. Results: Plasma Hcy in cases (11.3±5.3mmol/l) was significantly higher than in controls (8.3±5.0mmol/l, P<0.001). Hcy, TF, AT-III, prothrombin frgment 1+2, lipoprotein (a), diabetes, and smoking were significantly associated with young MI by using multiple logistic regression analysis (Table). In stepwise regression analysis, Hcy was the most important determinant of young MI (R^2=0.17, P<0.001). Hcy had significant positive correlation with TF (r=0.26, P=0.012) in cases but not in controls. Conclusion: These findings suggest that increased Hcy is an independent risk factor for young MI, mediated via activation of extrinsic coagulation.

	Odds ratio	95% Confidence Intervals	P value
Homocysteine	2.17	1.60-2.95	<0.001
Tissue Factor	1.56	1.18-2.06	0.002
Antithrombin	0.65	0.50-0.86	0.002
Fragment 1+2	1.45	1.11-1.90	0.007
Lipoprotein (a)	1.33	1.01-1.75	0.046
Diabetes	3.80	1.16-12.46	0.028
Smoking	3.12	1.18-8.25	0.022