

PE-053**The Use of Percutaneous Cardiopulmonary Support (PCPS) for Acute Myocardial Infarction (AMI)**

Kazuo Yamanaka
Ario Yamazato, Kazuhiko Doui, Akihiro Sugimoto
Takeda Hospital, Kyoto

Purpose: To determine the efficacy of PCPS for cardiogenic shock after AMI. **Patients and Methods:** In consecutive 81 patients who received PCPS in Takeda Hospital between January 1992 and August 2004, there were 50 patients (37males, 13females, average age, 68 years, age range, 47-87 years) with cardiogenic shock after AMI. 36 patients of them (72%) were used Intraaortic balloon pumping (IABP) at the same time. The induction of PCPS included before coronary angiography (18patients;36%), during coronary angiography (12patients;24%), low output syndrome after the operation for AMI (7patients;14%), mechanical complication after AMI (11patients;22%), and other (2patients;4%). Management of PCPS was conducted in accordance with the guidelines (Circ J 2002 ;66:133-144). **Results:** Mean PCPS time was 28.8 hours. 10 patients were transferred to the operating room under PCPS and performed an operation. 2 patients were died after the operation. In other 40 patients, 18 patients (45%) were weaned from PCPS. Mortality rate in patients with cardiogenic shock during percutaneous coronary intervention was especially high (78%). Total survived rate was 44%. Complication with PCPS system included bleeding at puncture site (48%), leg ischemia (16%), edema (14%), hematoma (12%) etc. **Conclusions:** Complications with PCPS system decreased, but survival rate was still low.

PE-054**Multivessel Disease Complicating Acute Myocardial Infarction Does Not Affect Left Ventricular Remodeling**

Noriaki Ito
Takakazu Morozumi, Shinsuke Nanto, Masaaki Uematsu, Jun-ichi Kotani, Masaki Awata, Toshinari Ohnishi, Osamu Iida, Fusako Oshima, Hitoshi Minamiguchi, Seiki Nagata
Kansai Rosai Hospital, Cardiovascular Division, Amagasaki

BACKGROUND: Although patients with multivessel disease (MVD) are associated with higher mortality rate in acute myocardial infarction (AMI), whether MVD affects left ventricular (LV) remodeling is unclear. **METHODS:** We retrospectively studied 100 consecutive patients with AMI (72 males, age ranged 38-84 years) who underwent primary angioplasty. Myocardial perfusion SPECT was serially performed two weeks after and six months after the procedure. Patients who demonstrated more than $\pm 30\%$ change in end diastolic volume index (LVEDVI) were excluded. Remaining 72 patients were classified into two groups: with single vessel disease (group S, N=19) and with MVD ($\geq 90\%$ stenosis, group M). Group M were subdivided into two groups: patients with complete revascularization by PCI in acute settings (M(+), N=11) and those underwent staged PCI (M(-), N=42). **RESULTS:** Patient demographic, peak CPK, peak CK-MB, defect extent score, severity score and LVEDVI two weeks after the onset (S vs M(+) vs M(-)= 57.5 ± 21.8 vs 62.7 ± 14.3 vs 62.0 ± 21.8 , n.s.) were all similar. % change in LVEDVI ($3.6 \pm 14.4\%$ vs $-5.1 \pm 17.4\%$ vs $-7.2 \pm 15.0\%$, $p < 0.02$; S vs M(+), $p < 0.03$; S vs M(-), $p < 0.02$) were different, but LVEDVI at six months after the procedure (58.5 ± 19.6 vs 58.1 ± 17.0 vs 57.4 ± 22.7 , n.s) were not different. **CONCLUSIONS:** Multivessel disease complicating AMI may not affect LV remodeling. Complete revascularization of MVD in acute settings failed to demonstrate beneficial effects on LV remodeling.

Heart Failure, Basic 3 (M)**PE10**

March 19 (Sat)

Poster Presentation Area (Exhibition Hall A+B+C)

9 : 55 – 10 : 40

PE-055**Periostin as a Anti-adhesion Molecule Responsible for Ventricular Dilation**

¹Yoshiaki Taniyama
¹Naruto Katsuragi, ²Toshio Ogihara, ¹Ryuichi Morishita
¹Division of Clinical Gene Therapy, Osaka University Graduate of Medicine, Suita,
²Department of Geriatric Medicine, Osaka University Graduate School of Medicine, Suita

Periostin is highly expressed in the myocardium in patients with heart failure. However, no report has documented the function of periostin. We investigated the function of periostin in vitro and checked whether loss of function of periostin can salvage cardiac heart failure in vivo. First, to address the molecular mechanisms of periostin, we employed the in vitro culture system. Especially, we focused on the effects of periostin on the cell attachment. We examined the attachment of cardiac fibroblasts on the plates coated with fibronectin, periostin and BSA. Interestingly, the cardiac fibroblasts did not attach to the plates coated with periostin, but not fibronectin or BSA $p < 0.01$. Second, to identify the function of periostin in the pathophysiology of heart failure, loss of function of periostin gene was examined using direct transfection into rat heart. We examined the inhibition of periostin in Dahl salt-sensitive rats by an antisense strategy, since periostin was highly expressed in heart failure. Importantly, inhibition of periostin gene expression by antisense ODN resulted in a significant increase in survival rate $p < 0.05$, accompanied by improvement of left ventricular function. Overall, the present study demonstrated that periostin inhibited cell adhesion which may cause cardiac dysfunction through the inhibition of the cell adhesion of cardiac fibroblasts. Inhibition of periostin might become a new therapeutic target for the treatment of heart failure.

PE-056**Morphological Basis for Impact of Angiotensin II Type 1A Receptor Blockade upon Myocardial Infarct Tissue Dynamics**

¹Yiwen Li
¹Genzou Takemura, ¹Hideshi Okada, ¹Shusaku Miyata,
¹Rumi Maruyama, ¹Masayasu Esaki, ¹Hirimitsu Kanamori,
¹Longhu Li, ¹Shinya Minatoguchi, ²Takako Fujiwara,
¹Hisayoshi Fujiwara
¹Department of Cardiology Gifu University School of Medicine, Gifu, ²Department of food science, Kyoto Woman's University, Kyoto

Blockade of angiotensin II type 1A receptor (AT1) attenuates left ventricular remodeling and heart failure after myocardial infarction (MI). However, the morphological bases for such beneficial effects are not fully elucidated except for prevention of fibrosis in the noninfarcted area. AT1 knockout mice (AT1KO), compared with the wild-type (WT), showed significantly attenuated left ventricular remodeling (dilatation) and dysfunction comprised of excessive fibrosis in noninfarcted area at 4 weeks after large MI, consistent with previous studies. Morphometry revealed that the size of infarct scar was comparable between each group, but that the infarct wall thickness was greater while its coronal expansion was less marked in AT1KO. The cell population in infarct scar was great in AT1KO, where extravascular alpha-smooth muscle actin (SMA)-positive cells and vessels was abundant. In 1-week-old infarct consisting of granulation tissue, apoptosis of alpha-SMA-positive cells and endothelial cells was less frequent in AT1KO than in WT. Thus, in AT1KO, the cells escaped from apoptosis might have contributed to abundant cell population and infarct wall thickening, which consequently altered the infarcted tissue geometry to reduce wall stress, and then prevented left ventricular dilatation and dysfunction at the chronic stage. Unfavorable infarct tissue dynamics, in addition to fibrosis, may be an important mechanistic basis for deleterious effects of AT1 signals upon the postinfarction disease process.