

e0013 MATRIX METALLOPROTEINASES-9—A BIOMARKER IN AORTIC DISSECTION

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Aortic dissection (AD) is a life threatening emergency. Early diagnosis and treatment are pivotal for patients with AD. Matrix metalloproteinases (MMPs), through degradation of extracellular matrix, may play an important role in these processes. Elevation of MMPs might represent an opportunity to diagnostically characterise acute aortic processes. We examined the potential diagnostic role of MMP-9 in AD.

Methods Between February 2009 and November 2009, 86 consecutive patients affected by acute AD who were admitted to our emergency department for evaluation were enrolled in this study (Traumatic AD, Marfan Syndrome were excluded). Blood samples were obtained from venipuncture in EDTA tubes from fasting patients at the time of hospital admission. Plasma levels of MMPs were evaluated by ELISA in 86 patients affected by AD (28 type A, 58 type B). 30 healthy subjects were used as controls. Aortic tissue samples obtained during surgery were evaluated by immunohistochemistry and western blot for MMP-9 expression. In addition, control tissue was obtained from subjects who underwent heart transplantation (2 male and 1 female). Data were analysed by SPSS statistical software (version 13.0). All data were presented as mean±SEM. A two-tailed unpaired Student t test was used to assess differences in MMP levels. $p<0.05$ was considered statistically significant.

Results Mean plasma values for MMP-9 were higher in patients affected by AD compared to control group at the time of hospital admission (22.78 ± 1.28 ng/ml vs 17.45 ± 1.36 ng/ml $p<0.05$). Moderate/strong expression (+++++) of MMP-9 was present in both the intima and media layers at the entry site in AD compared to control aortas, Western blot analysis confirmed the expression of MMP-9 in AD whereas no/low (-/+) expression was present in control aortas.

Conclusions This pilot study suggests that MMPs play an important role in the pathophysiology of AD and that the acute phase of AD is characterised by an increase of MMP-9 plasma levels. In this setting, the detection of aortic dissection using biochemical techniques would represent an attractive, rapid, non-invasive and easy to perform diagnostic tool. Future investigation of MMPs and their inhibitors in AD will likely lead to advancements in the diagnosis, prognosis, and treatment of this deadly disease.

e0014 THE ROLE OF PEROXISOME PROLIFERATORACTIVATED RECEPTOR COACTIVATOR 1 IN MYOCARDIAL ISCHAEMIC PRECONDITIONING AND DIAZOXIDE PRECONDITIONING

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Objective Ischemic preconditioning (IPC) is an important endogenous protective mechanism for ischaemia-reperfusion (I/R) injury. Currently, it is believed that opening of the mitochondrial ATP-sensitive potassium channel (mitoKATP channel) plays an important role in IPC. Diazoxide is considered as a mitoKATP agonist that is capable of activating endogenous protective mechanism of the myocardium to antagonise myocardial ischaemic and hypoxic injury. In this study, we applied a rat isolated perfused heart model to observe the expression characteristics of peroxisome proliferator-activated receptor γ coactivator-1 α (PGC-1 α) and ultrastructural

changes of mitochondria after IPC and diazoxide preconditioning (DPC), in order to explore the protective role and mechanism of IPC and DPC during IR injury of the heart.

Methods 30 Wistar rats were used to establish Langendorff isolated perfused heart model. Rats were randomly divided into five groups, six in each group: (1) I/R group: after 30 min of equilibration perfusion, heart was subjected to 30 min of ischaemia and 1 h of reperfusion. (2) IPC group: after 10 min of equilibration perfusion, heart was subjected to two times of 5 min ischaemia and 5 min reperfusion, followed by a 30 min of ischaemia and 1 h of reperfusion. (3) DPC group: after 10 min of equilibration perfusion, heart was given two times of K-H perfusion solution containing diazoxide (100 μ mol/l) for 5 min then non-diazoxide K-H perfusion solution for 5 min, followed by 30 min of ischaemia and 1 h of reperfusion. (4) Blank control group: equal amount of saline was used instead of diazoxide. Perfusion procedure was the same as the DPC group. (5) Dimethyl sulfoxide (DMSO) group: DMSO was applied instead of diazoxide, and perfusion procedure was the same as the DPC group. Cardiac apex muscle was cut for frozen section. Immunohistochemistry staining of PGC-1 α was performed and average absorbance was calculated. Electron microscope was used for Flameng scoring of the myocardial mitochondria.

Results The average absorbance values of PGC-1 α were respectively: I/R group (3.88 ± 1.72), IPC group (10.94 ± 5.23), DPC group (8.40 ± 3.64), blank control group (3.55 ± 1.56) and DMSO group (4.16 ± 0.52). The expression of PGC-1 α was significantly increased in IPC and DPC groups and the differences were statistically significant compared to the I/R, blank control and DMSO groups, that is, $p<0.01$ for IPC group and $p<0.05$ for DPC group. However, there was no significant difference between the IPC and DPC groups ($p>0.05$). Flameng score: IPC group (0.44 ± 0.13), DPC group (0.47 ± 0.10), I/R group (1.78 ± 0.14), blank control group (1.70 ± 0.03) and DMSO group (1.68 ± 0.06). Flameng score of the IPC and DPC groups had statistically significant difference as compared to the I/R group, blank control group and DMSO group ($p<0.01$), but no significant difference was detected between the IPC and DPC groups ($p>0.05$).

Conclusion IPC and DPC have a protective effect on myocardial mitochondria, and their mechanism in action may be related to activation and over-expression of PGC-1 α .

e0015 EFFECT OF THE ISCHAEMIC PRECONDITIONING ON ISCHAEMIC REPERFUSED MYOCARDIUM OF ELDERLY RAT HEART

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Objective To explore the effect of ischaemic preconditioning (IPC) on ischaemic-reperfusion (I/R) myocardium of elderly rat.

Methods 56 male Wistar rats were divided into seven groups of eight rats each: adult control group, adult I/R group, adult IPC group, elderly control group, elderly I/R group, elderly IPC group and elderly enhanced IPC group.

The Langendorff isolated heart perfusion models were established. The control group had a 90-min perfusion without any intervention. The I/R group had a 30-min equilibration period and a 30-min ischaemia and a 30-min reperfusion. The IPC group had a 10-min equilibration, then was elicited by two cycles of 5 min of ischaemia interspersed with 5 min reperfusion prior to 30 min ischaemia and a 30-min reperfusion. The enhanced IPC group had a 10-min equilibration, then was elicited by four cycles of 5 min of ischaemia interspersed with 5 min reperfusion prior to 30 min ischaemia and a 30-min reperfusion. The recovery rate of the left ventricular function, such as cardiac output (CO), left ventricular