Coronary Intervention Endothelial Function and Natriuretic Peptide System

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Background: Flow mediated dilatation (FMD) reflects endothelial function. Adequate levels of endothelial cGMP are necessary for FMD. eNOS , (a NO activation of soluble Guanilate Cyclase (GC)) and Corin (natriuretic peptide activation of membrane GC) contribute to cGMP levels. ED described post-PCI, is probably mediated by contrast media (CM) and can be reversed by human recombinant (hr) BNP treatment.

Aims: To evaluate whether ED post PCI can be prevented by hrBNP and to investigate the underlying mechanism.

Methods and results: FMD (antecubital ultrasound), blood Corin and BNP levels (ELISA) were measured before, and 24 hours post-PCI in 111 patients. ED was defined as > 2.5% loss of baseline FMD. The patients were randomized to two groups: the “control" (n=67) received nitroglycerin and the "BNP" (n=44) received hrBNP during their PCI. Human cardiac microvascular endothelial cells (HCMEC) were exposed to CM with and without BNP. Cellular Corin, eNOS and cGMP were measured (ELISA).
ED post-PCI developed in 62% of the "control", as compared to 42.1% of the "BNP" (p=0.04). Corin (780.78 ± 317.87 pg/ml to 833.13 ± 321.04, p=0.003) and BNP (94.5 ± 137.75 pg/ml to 224.69 ± 225.97, p<0.001) levels increased significantly in the "control", but not in the "BNP" (801.04 ± 250.64 pg/ml to 827.83 ± 255.48, p= 0.076, and 119.69 ± 171.82 pg/ml to 149.68 ± 201.67, p= 0.096).
HCMEC: CM reduced eNOS (1280± 21pg/106 cells in the control to 962 ± 12 ,p=0.001) increased Corin (1330± 14 pg/ml in the control to 1661±25 pg/ml/106 cells, p=0.002), and slightly reduced cGMP (186±8 pg/ml/106 cells vs.195± 16, p=0.278). CM +BNP: Similar changes in eNOS and Corin levels were noted (-23% and +22%), with cGMP increasing to 258 ±8 pg/ml/106 cells, p<0.001).

Conclusions: CM induced ED results from impaired eNOS expression. A compensatory rise in Corin along with administration of BNP, probably maintains cytosolic cGMP levels to prevent ED.