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MEETING ABSTRACT Open Access

Arterial stiffness and collagen expression in mice lacking NO-sensitive guanylyl cyclase

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Background

NO-sensitive guanylyl cyclase (NO-GC) is the most important receptor for NO. The NO signal is transmitted through an increase in cGMP to act on several effector molecules. The NO/cGMP cascade is involved in the regulation of vessel diameter ultimately contributing to set the blood pressure. Lack of NO or NO-GC is thought to alter the responsiveness of blood vessels as well as their composition. We hypothesize that arterial stiffness may be altered in the absence of NO-GC.

Methods

As arterial stiffness is an import indicator of cardiovascular risks and mortality, we aimed at determining the role of NO-GC for aortic stiffness using general KO mice (GCKO) and smooth muscle-specific KO mice (SMC-GCKO) for NO-GC. Measurement of pulse wave velocity (PWV) was frequently used to determine aortic stiffness. We used magnetic resonance tomography (17.6 Tesla) for non-invasive measurement of local aortic PWV. PWV was calculated by simultaneously measuring of cross-sectional change and volume flow in a defined time.

Results

Preliminary data indicate an increased PWV in GCKO mice whereas in SMC-GCKO, stiffness is unaltered. Aortic diameter was reduced in GCKO compared to WT whereas that in SMC-GCKO was not significantly different from controls. Global deletion of NO-GC did not affect cardiac output. These data will be correlated to structural data of aorta from these strains.

Conclusion

Deletion of NO-GC affects the physical properties of vascular tissue. This effect appears to be independent of hypertension.

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