Changes of thoracic and mesenteric arterial vascular function in angiotensin Ⅱ-induced hypertensive mice

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Endothelial cells play an important role in regulation of vascular function, which are affected by various bioactive substances. Angiotensin (Ang) II induces vasoconstriction and is suggested to cause vascular dysfunction. In this study, we compared the endothelium-dependent vasorelaxation response of the thoracic aorta and mesenteric artery in Ang II -induced hypertensive model mice.

In thoracic aorta, stimulation of endothelial a_2-adrenoceptor with clonidine induced a PI3-K-dependent relaxation, whereas acetylcholine (ACh) induced the relaxation via Gq-PLC-Ca^{2+} pathway. In mesenteric artery, ACh-induce relaxation was remarkable, but clonidine-induced relaxation was hardly observed. Ang II -induced hypertension significantly impaired the endothelium-dependent relaxation in aorta, but not in mesenteric artery. Experiment using NOS inhibitor revealed that vasorelaxation responses to clonidine and ACh in aorta were largely dependent on NOS activity, whereas NO played minor role in endothelial-dependent vasorelaxation in the mesenteric artery. These results suggest that NOS-dependent endothelial regulation of vascular tone is more sensitive to Ang II -induced vascular dysfunction. In addition, the difference in endothelium-dependent relaxation mechanism may be related to the susceptibility of distinct blood vessel to Ang II -induced vascular injury.