Abstract

Background: Vasohibin-2 (VASH2) has been isolated as a homologue of vasohibin-1 (VASH1) that promote angiogenesis counteracting with VASH1. Chronic angiotensin II (AngII) infusion promotes both ascending and abdominal aortic aneurysms (AAs) in mice. The present study aimed to investigate whether exogenous VASH2 influenced AngII-induced vascular pathology in apolipoprotein E deficient (*ApoE*$^{-/-}$) mice.

Methods: Male, *ApoE*$^{-/-}$ mice (9 to 14 weeks old) were injected with Ad LacZ or Ad VASH2. After a week, saline or AngII (1,000 ng/kg/min) was infused into the mice subcutaneously via mini-osmotic pumps for 3 weeks. Consequently, all these mice were divided into 4 groups: saline + LacZ (n=5), saline + VASH2 (n=5), AngII + LacZ (n=18), and AngII + VASH2 (n=17).

Results: Exogenous VASH2 had no significant effect on *ex vivo* maximal diameters of abdominal aortas (AngII + LacZ; 1.67±0.17 mm, AngII + VASH2; 1.52±0.16 mm, n.s.) or elastin fragmentation and accumulation of inflammatory cells. Conversely, exogenous VASH2 significantly increased intima areas of aortic arches (AngII + LacZ; 16.6±0.27 mm^2, AngII + VASH2; 18.6±0.64 mm^2, p=0.006). VASH2 effect of AngII-induced ascending AAs was associated with

increased cleaved caspase-3 abundance. AngII-induced atherosclerosis was not altered by VASH2.

Conclusion: The present study demonstrated that augmented VASH2 expression had no effect of AngII-induced abdominal AAs or atherosclerosis, while increasing dilation in the ascending aorta.