Correspondence

Letter by Bi et al Regarding Article, “Segmental Aortic Stiffening Contributes to Experimental Abdominal Aortic Aneurysm Development”

To the Editor:

We read with great interest the important article by Raaz and colleagues, who demonstrate a causal effect of segmental aortic stiffness in abdominal aortic aneurysm (AAA) formation. This article successfully shows how AAA initiation seems to be a story of the butterfly effect. Previously, we introduced a novel experimental model of enlarging AAA in rabbits by a combination of elastase incubation and aortic coarctation. We hypothesized that hemodynamic change caused by coarctation may play an essential role in the initiation and progression of AAA. In comparison, Raaz and colleagues apply an integrated biophysical approach to study AAA. In their study, segmental aortic stiffness generates axial wall stress during systolic aortic expansion and increases the expression of genes related to inflammation and extracellular matrix remodeling. The authors deliberately chose the porcine pancreatic elastase model and claimed that this model is also initiated by mild destruction of the elastin. However, in a rat model, Yamaguchi and colleagues demonstrated AAA formation caused by exogenously infused elastase and elastin degeneration immediately after elastase infusion. In our rabbit study, AAA did not form unless the aorta was degenerated dramatically by high concentrations of elastase.

These fast-induced models are quite different from human AAA, and the self-healing period is the main limitation. Raaz and colleagues also found decelerating aortic diameter enlargement after day 7, and attributed this to a decline in segmental aortic stiffness. In our study, we proposed a self-healing process whereby regenerated elastin and proliferated smooth muscle cells inhibit aneurysm enlargement further. We questioned the classical theory that aneurysms develop after infusion of exogenous elastase resulting in an inflammatory cascade, which causes matrix destruction and aneurysm formation by matrix metalloproteinases (MMPs). According to most theories, it seems that MMPs are evil, and studies try to break this vicious circle by inhibiting MMPs. Conversely, we appreciate that aneurysm growth is an active process, as opposed to simple passive dilation, and hypothesize that MMPs play a vital role, even positive role, in the complex repair process of the aneurysm. Shen and colleagues reported that MMP2 plays 2 opposing roles in aortic wall remodeling. Endogenous MMPs, especially MMP2, should be a key target for investigation and treatment of aortic aneurysm.

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Disclosures

None.

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