Role of Microvascular Tone and Extracellular Matrix Contraction in the Regulation of Interstitial FluidHighlights

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Résumé en anglais	The pathophysiology of aortic dissection is poorly understood, and its risk is resistant to medical treatment. Most studies have focused on a proposed pathogenic role of transforming growth factor-β in Marfan disease and related thoracic aortic aneurysms and aortic dissections. However, clinical testing of this concept using angiotensin II type 1 receptor antagonists to block transforming growth factor-β signaling fell short of promise. Genetic mutations that predispose to thoracic aortic aneurysms and aortic dissections affect components of the extracellular matrix and proteins involved in cellular force generation. Thus, a role for dysfunctional mechanosensing in abnormal aortic wall remodeling is emerging. However, how abnormal mechanosensing leads to aortic dissection remains a mystery. Here, we review current knowledge about the regulation of interstitial fluid dynamics and myogenic tone and propose that alteration in contractile force reduces vascular tone in the microcirculation (here, aortic vasa vasorum) and leads to elevations of blood flow, transmural pressure, and fluid flux into the surrounding aortic media. Furthermore, reduced contractile force in medial smooth muscle cells coupled with alteration of structural components of the extracellular matrix limits extracellular matrix contraction, further promoting the formation of intramural edema, a critical step in the initiation of aortic dissection. The concept is supported by several pathophysiological and clinical observations. A direct implication of this concept is that drugs that lower blood pressure and limit interstitial fluid accumulation while preserving or increasing microvascular tone would limit the risk of dissection. In contrast, drugs that substantially lower microvascular tone would be ineffective or may accelerate the disease and precipitate aortic dissection.

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