Insights into aortic remodeling in type B aortic dissection using patient-specific fluid-structure interaction simulations

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Abstract

Aortic dissection, a life-threatening condition, arises from the sudden creation of a parallel flow channel within the delaminated aortic wall, known as the false lumen (1). In its chronic phase, the primary drivers of late complications are the degeneration and dilatation of the false lumen. The complex interplay between hemodynamics and microstructural remodeling, which leads to extensive anatomical remodeling over the course of years, is not yet fully understood.

This study (2) examines the progression of a patient's aortic dissection, captured by surveillance imaging with CT angiography from pre-dissection to the chronic phase over a total period of seven years. We applied monolithically coupled fluid-structure interaction models with tissue prestress, external tissue support, and an anisotropic tissue model to identify potential growth-related hemodynamic markers. In the aortic wall domain, we independently assigned thickness and material properties to each functional tissue layer. In vitro 4D-flow MR imaging (3) and the patient's blood pressure were used to inform the boundary conditions.

Quantitative measurements during routine clinical care showed that aortic dilatation was most significant distal to the left subclavian artery, reaching 6 cm in the chronic phase. Simulations resulted in a flow jet velocity through the entry tear that peaked in the subacute phase and successively decreased in the chronic phase, corresponding to an increased entry tear size. Flow jet impingement on the false lumen resulted in a localized increase in pressure and wall shear stress in the subacute and chronic phases. These hemodynamic changes appear to be the main drivers of aortic growth and morphological changes. Despite moderate overall flap movement, in-plane displacement increased as disease progressed, which was associated with an overall increase in aortic diameter. In fact, additional simulations with a significant reduction in flap stiffness during the subacute phase resulted in increased flap motion up to 9.5 mm. Although these results are based on a single patient, they suggest a strong relationship between hemodynamics and aortic growth.

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