Experimental characterization of aneurysmal aortic tissue rupture under bulge inflation loading in a confocal microscope

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ABSTRACT

The wall of the ascending thoracic aorta shall withstand, absorb and transform massive systolic ejection energy at each cardiac cycle. To fulfill this role, it retains a complex heterogeneous microstructural organization. Ascending thoracic aortic aneurysms (ATAAs) are associated with a degenerative remodeling process of this microstructure, which results in an altered mechanical behavior. The deformation micro-mechanisms which drive the macro-mechanical response and can potentially trigger a localized rupture in ATAAs represent an extremely relevant issue that is currently unidentified. The presented approach combined bulge inflation tests on human ATAA specimens and 3D *in situ* observations of their fibrous microstructure while inflating. The tests performed on 13 specimens included two steps. The first one ranged from unloaded to systolic load states, targeting the (non-destructive) macro-mechanical elastic characterization through stereo digital image correlation analysis and inverse identification of the specimens' mechanical properties [1]. The second one ranged from unloaded up to rupture state, with the goal of obtaining a 3D *in situ* microstructural characterization of the adventitial fibrous structures through multiphoton confocal microscopy and quantitative image analysis [2]. Adventitial collagen end elastic fibers within the adventitia were continuously observed and the evolution of their straightness and orientation dispersion was assessed. Furthermore, microstructural and macro-mechanical parameters were compared to morphometrical parameters like aneurysm diameter and wall thickness.

Our results confirmed the recruitment process of adventitial collagen and elastin fibers at physiological loads. They also emphasized the complexity and heterogeneity of the deformation mechanisms at higher loads. Collagen fiber bundles were found to undergo decohesion at supra-physiological pressures preceding rupture. In addition, our observations suggested the nucleation and growth of pores within the tissue long before the rupture, and the likely propagation of the rupture from inner medial layers. Wall thickness and aneurysm diameter demonstrated similar associations to microstructural parameters.

REFERENCES

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