PATIENT-SPECIFIC FINITE ELEMENT MODELLING OF INTRACRANIAL ANEURYSM GROWTH AND RUPTURE

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Abstract

Intracranial aneurysms (IAs) are localized dilations of cerebral arteries that pose significant clinical risks, particularly when ruptured, causing subarachnoid haemorrhage (SAH). Unruptured IAs affect 2–5% of the population, with risks influenced by factors like age, gender, genetics, hypertension, and smoking (1,2). During IA evolution, significant extracellular matrix (ECM) changes occur, including elastin degradation, collagen remodelling, and loss of smooth muscle cells (SMCs), leading to reduced vessel wall strength and stiffness (3). Despite advancements in imaging techniques, rupture risk assessments largely rely on qualitative metrics and clinician judgment lacking integration of patient-specific variations in aneurysm morphology, hemodynamic, and vessel wall biology. Patient-specific models aim to address these gaps by incorporating individualized data, such as vessel geometry and tissue remodelling, to predict IA progression. In the current study we propose a framework to predict arterial growth based on cell level remodelling, leading to a diagnostic tool to predict patient-specific IA growth. A constitutive law is implemented to describe elastin pre-tension, anisotropic collagen strain stiffening, and active smooth muscle cell (SMC) contractility (4). The active cell free energy and passive ECM elastic energy is computed and cell and tissue remodelling is initiated when the total artery free energy changes significantly from the homeostatic free energy of a healthy artery. The initiation and evolution of fusiform and saccular IA geometries are simulated. A patient specific geometry of the internal carotid artery is reconstructed using image segmentation with SimVascular, and a finite element mesh of the vessel is created using the GIBBON toolbox. Our active anisotropic artery constitutive law and our thermodynamically based growth model are implemented in a user-defined material subroutine (UMAT) in the Abaqus implicit finite element solver. Steady state conditions for artery homeostasis are predicted for a healthy vessel at 120 mmHg. An initial parametric study investigates the effect of elastin degradation on the vessel radius in the absence of growth and remodelling, revealing that full disruption of elastin results in merely a 14% increase in vessel radius. This suggests that elastin degradation acts as an initiation trigger for IA formation, but does not account for large radius increases observed for fully developed aneurysms. We then simulate the effects of growth in a vessel experiencing hypertensive conditions and elastin degradation. An increase in free energy due to increased stretching of the passive ECM results in initial growth. Following this, SMCs are predicted to transform from a contractile phenotype to a synthetic phenotype, further increasing the free energy of the system and significantly increasing the growth of the IA. We demonstrate the ability of the model to predict both saccular and fusiform IA development, depending on the localisation of elastin damage during initial stages of IA evolution. A cohesive zone formulation is used

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to predict the rupture risk for the IA, with the cohesive strength dependent on the predicted evolution of collagen concentration and alignment. Results are compared to clinical data for IA evolution and rupture.

References
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