The Effect of Lipid Core Position on Carotid Fibrous Cap Stress Levels

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Introduction

Atherosclerosis is the main cause of death and severe disability in the world. The disease generates atherosclerotic plaques consisting of lipid cores covered by a protective fibrous cap. Rupture of the fibrous cap forms blood clots which may be carried down-stream to cause strokes in the case of carotid plaques. Currently the risk of cap rupture is assessed using the degree of luminal narrowing which fails to take the morphology of the plaque into account. Indeed, unstable or vulnerable plaques are known to possess large lipid cores and thin fibrous caps (1). High levels of first principal stresses in the carotid artery plaque are hypothesized to induce plaque rupture (2). We sought to investigate if the intraplaque position of the lipid core would influence stress levels in the fibrous cap, and thereby affect plaque vulnerability. We used fluid structure interaction analysis based on magnetic resonance imaging (MRI) of the carotid artery and simulations were performed on three models with varying intraplaque lipid core positions.

Methods

MR data acquisition and segmentation

A patient awaiting operation for severe carotid plaque was scanned using a well-validated MRI protocol (3). Sixteen transverse slices were scanned using cardiac-gated turbo spin echo sequences: T1W (TR/TE/Inv: 1RR/8/650 ms), T2W and PDW scans with TR=3RR and TE=40/20 ms, and a TOF sequence (TR/TE 34.9/2.4 ms). All four sequences covered a field of view of 16x12 cm using a 256x256 matrix yielding a raw resolution of 0.61x0.61mm with 2 mm slice thicknesses. The image intensities of each scan compared to the adjacent sternocleid muscle were analyzed using the software Cascade, a dedicated automated segmentation tool, allowing segmentation into lipid core, fibrous cap, vessel wall, and blood stream (4). Velocities were measured 2 cm up- and downstream from the flow divider using phase-contrast imaging (Venc=150 cm/s).

Model generation and fluid structure interaction analysis

Each segmented slice was imported into Matlab R2007a to generate a 3D shell model using linear interpolation and gaussian smoothing between slices in order to derive an isotropic dataset. To provide a 2D longitudinal model transecting the center of the bloodstream, the 3D shell model was intersected by a curved surface coincident with points located central to the bloodstream in the three carotid segments. This 2D longitudinal model was used as a template for three models created with a high-grade stenosis of 90% (measured according to the ECST standard (5)) and a proximal, central, and distal lipid core position (figure 1). Minimal fibrous cap thickness was 0.5 mm in all the models. Blood flow was simulated as an incompressible homogenous Newtonian fluid (ρ=1050, v=0.005). Laminar outflow profiles were specified at the internal and external carotids using the mean velocity measured at the time of maximal flow. Pressure in the common carotid was set to the systolic blood pressure of the patient (160 mmHg). A hyper-elastic model was used to specify the material properties of the surrounding tissues. A fluid structure interaction plane strain simulation was performed using Comsol Multiphysics 3.4 coupling the flow induced deformations to stresses in the tissues surrounding the carotid vessel.

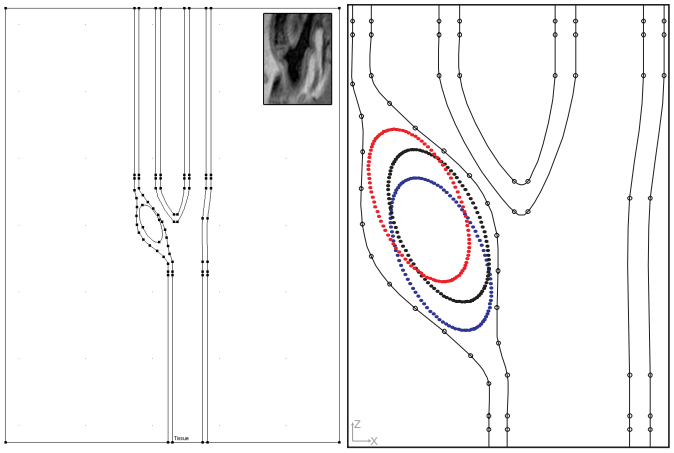

Figure 1: Left: Model simulated. Inset shows a longitudinal black-blood MRI image of the carotid bifurcation. Right: Three models were simulated with varying lipid core placement. The proximal/distal models are mirror images of one another around the short axis of the plaque. The central model is symmetrical around the same axis.

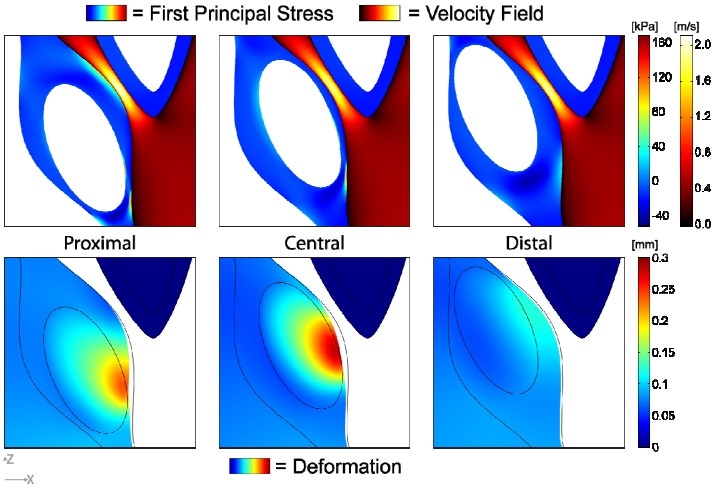

Figure 2: Main results. First principal stresses and velocity fields are depicted (top). Deformations caused by the flowing blood are depicted (bottom).

Results

The maximal stresses occurred in the transitional region of high to low deformation at the shoulder regions of the plaque (figure 2). The position of the lipid core affected first principal stress levels in the fibrous cap. The maximal stresses with the lipid core positioned in the proximal, central, and distal portion of the plaque were 131 kPa, 87 kPa, and 98 kPa, respectively.

Discussion

First principal stress levels were highest with a proximal position of the lipid core in our simulations. This may explain the findings of Lovett et al (6), that carotid plaques tend to rupture upstream (proximally) rather than downstream (distally). Furthermore, even though the model with the lipid core in the center position deformed more than the other two models, maximal first principal stress levels were the lowest of all the models. Lipid cores placed closer to the shoulder region of the plaque gave rise to significantly greater stresses, providing a biomechanical explanation for the histological finding, that plaques tend to rupture at the shoulder regions. Computational analyses may yield valuable additional information concerning fibrous cap stress levels which may support current methods of diagnostics.

References

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