Longitudinal 2D Stresses in Carotid Plaques using MRI-based Fluid Structure Interaction Models

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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). This morphology generates severe internal stresses in the fibrous cap. In vitro studies have shown that cap rupture predominantly occurs when static stresses exceed 300 kPa(2). The ability to estimate stress magnitudes in the fibrous cap is thus expected to improve risk assessment.

Methods

MR data acquisition

Patients awaiting operation for severe carotid plaque were 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) (figure 1). All four sequences covered a field of view of 16x12 cm using a 256x256 matrix resulting in a raw resolution of 0.61x0.61mm with 2mm slice thickness. The image intensities of each scan compared to the adjacent sternocleid muscle were analyzed using 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). To resolve the motion of the vessel walls, dynamic B-TFE scans with 20 cardiac phases were performed using a single longitudinal slice and five transverse slices (TR/TE/slice thickness/inplane resolution: 5.7ms/2.8ms/8mm/0.74x0.76mm and 7.7ms/3.9ms/2mm/0.64x0.52mm, respectively).

Segmentation and fluid structure interaction analysis

Each segmented slice was imported into Matlab to generate a 3D 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 throughout the model, the 3D model was intersected using a NURBS surface created from user-selected skeletonization points (figure 2). Blood-flow was simulated as an incompressible homogenous Newtonian fluid (ρ=1050, ν=0.005) (COMSOL Multiphysics, COMSOL AB, Stockholm, Sweden). A parabolic inflow profile was specified using the maximal central velocity measured at the time of maximal flow. Pressure in the internal and external carotid outlets was set to the systolic blood pressure. A Neo-Hookean hyper-elastic model was used to specify the material properties of surrounding tissue (μ=6.20e6, κ=1.24e8, ρ=960) and vessel wall (μ=7.20e5, κ=1.44e7, ρ=1200). Lipid was treated as an isotropic materiel with Young's modulus set to 1/100th of the vessel wall (E=1e5, ν=0.45, ρ=900). Pressure and viscous forces were used to couple the fluid forces to the structural deformation in the surrounding vessel wall components along the vessel-wall/blood-stream interface.

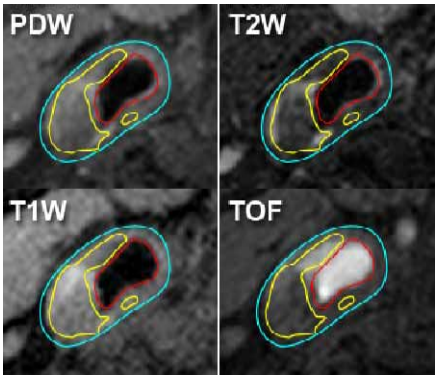

Figure 1: *Segmentation results from the multi-contrast weighted MRI scans.*

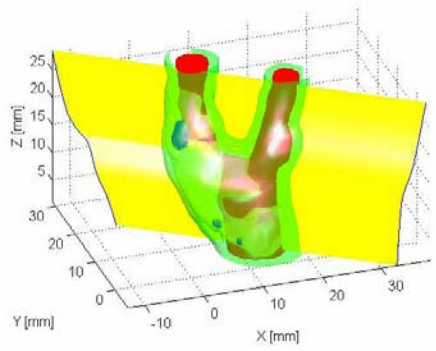

Figure 2: *Intersection of the blood-stream by a NURBS surface calculated from user-selected skeletonization points*

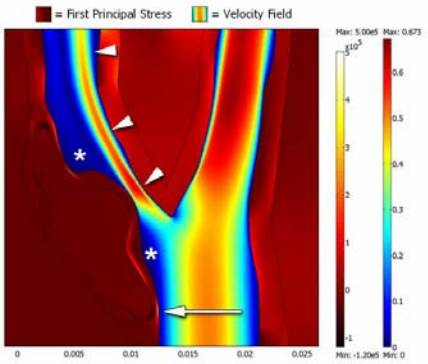

Figure 3: *Main results: fibrous cap exhibits severe stresses, maximal at inlet shoulder (arrow). Luminal narrowing results in velocity jet (arrowheads) + areas of recirculating blood (*)*

Results

First principal stresses of the vessel wall and fibrous cap as well as velocity field and pressure of the bloodstream were calculated (figure 3). The fibrous cap was defined as the part of the vessel wall overlaying the lipid pool. This region was exposed to the greatest principal stresses as a result of the marked deformation of this region due to the underlying soft lipid pool. The stresses were maximal in the inflow "shoulder region" i.e. the region of the fibrous cap adjacent to the vessel wall. Stagnation points occurred at the flow divider. The severe luminal narrowing generated marked velocity jets and large areas of recirculating or slowly moving blood, a known progenitor of further plaque deposition.

Discussion

The stresses found in the fibrous cap approach established criteria for caps at risk of rupture(2). Maximal stresses occurred in the inflow shoulder region i.e. the section of plaque adjacent to the vessel wall (arrow, figure 3), the preferential site of plaque rupture. Our results are thus in agreement with clinical findings of plaque physiology. Future comparisons of mechanical stresses matched to plaque histology may provide new insights into the etiology of vulnerable plaques and ultimately lead to improved risk stratification of patients by incorporating fibrous cap stresses in the risk assessment.

References

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