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Effects of varied lipid core volume and fibrous cap thickness on stress distribution in carotid arterial plaques

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ABSTRACT

The rupture of atherosclerotic plaques is known to be associated with the stresses that act on or within the arterial wall. The extreme wall tensile stress is usually recognized as a primary trigger for the rupture of the plaque. The present study used one-way fluid–structure interaction simulation to investigate the impacts of fibrous cap thickness and lipid core volume to the wall tensile stress value and distributions on the fibrous cap. Von Mises stress was employed to represent the wall tensile stress (VWTS). A total of 13 carotid bifurcation cases were manipulated based on a base geometry in the study with varied combinations of fibrous cap thickness and lipid core volume in the plaque. Values of maximum VWTS and a stress value of VWTS_90, which represents the cut-off VWTS value of 90% in cumulative histogram of VWTS possessed at the computational nodes on the luminal surface of fibrous cap, were used to assess the risk of plaque rupture for each case. Both parameters are capable of separating the simulation cases into vulnerable and more stable plaque groups, while VWTS_90 is more robust for plaque rupture risk assessment. The results show that the stress level on the fibrous cap is much more sensitive to the changes in the fibrous cap thickness than the lipid core volume. A slight decrease of cap thickness can cause a significant increase of stress. For all simulation cases, high VWTS appears at the fibrous cap near the lipid core (plaque shoulder) regions.

1. Introduction

Atherosclerotic cardiovascular disease is one of the leading causes of death in western countries (Petersen et al., 2005). More than 60% of myocardial infarction is caused by the rupture of a vulnerable plaque (Casscells et al., 2003). It is widely accepted that a plaque with a large, soft, lipid-rich necrotic core, and covered by a thin fibrous cap is more vulnerable to rupture (Davies and Thomas, 1985). Rupture of this thin cap and then the subsequent thrombus formation are thought to be the most important mechanism underlying acute coronary syndromes and stroke. The rupture of the fibrous cap depends not only on its biological features (Burke et al., 1997; Falk, 1992; MacIsaac et al., 1993), such as inflammation, but also on the biomechanical factors, including the wall shear stress (WSS), wall tensile stress, and cyclic force caused by the pulsatile blood pressure (Richardson et al., 1989; Van der Wal and Becker, 1999; Lee, 2000).

Structural analyses of the stress in the coronary and carotid arteries with plaque have been developed by many researchers from 2D to 3D models, from structure analysis to the fluid–structure interaction (FSI) models. Early researches on the plaque rupture risk assessment were mainly on the 2D models (Loree et al., 1992; Chenu et al., 1993; Lee et al., 1993, 2000; Huang et al., 2001). Based on 2D histological models, Huang et al. (2001) found that thin fibrous cap with a large lipid core are important determinants of increased plaque stress. Cheng et al. (1993) found that a circumferential stress value of 300 kPa could be used to predict whether the plaque is stable or unstable based on a 2D finite element study on ruptured- and un-ruptured plaques in human coronary arterial specimens. Also on 2D models, Loree et al. (1992) found that reducing the fibrous cap thickness dramatically increases peak circumferential stress; Tang et al. (2004a) investigated the influence of the fibrous cap thickness and the lipid core size on the stress distribution on 2D models, later they studied the stress change on track points in the fibrous cap with 3D FSI models under varied fibrous cap thickness (Tang et al., 2005). So far, there is no systematic study on the influence of gradually varied lipid core volume and fibrous cap thickness to the stress distribution changes in 3D carotid bifurcation—one of the most important sites on plaque rupture.

In the present study, based on realistic multi-component arterial plaque geometry, a total of 13 carotid bifurcation models were manipulated with varied combinations of fibrous cap thickness and lipid core volume but the same degree of stenosis.

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One-way FSI simulations were performed on the cases to investigate the impacts of the specific combination of fibrous cap thickness and lipid core volume to the stress distribution.

2. Methodology

2.1. Model geometry reconstruction

The detailed description of 3D arterial plaque reconstruction from histology sections can be found in the Appendix A. The base model shown in Fig. 1(a), which was reconstructed from histology images, was used in the study as Case 2. Based on this model, 12 cases were manipulated, with fixed arterial wall inner and outer surfaces but varied lipid core volumes and locations to provide different combinations of fibrous cap thickness and lipid core volume (Fig. 1(b)). Table 1 presents the study case matrix. For the cases with the same fibrous cap thickness, the surface of lipid core at the luminal side was fixed, the rest parts were moved proportionally to change lipid core volume as shown in Fig. 1(b) "i" from Cases 2 to 4; for the cases with the same lipid core volume and different cap thickness, the surfaces of lipid core was moved proportionally towards or away from the luminal surface, as shown in Fig. 1(b) "ii" from Cases 2 to 11. The shape of the fibrous cap will remain similar for the cases through the manipulation procedure, while fibrous cap thickness will not be the same throughout the plaque for the same case. The minimum value of the fibrous cap thickness for each case is presented here. This manipulation process is purely for simulation purpose of creating geometry models with varied cap thickness or lipid core volume. It does not mean that the plaque will grow in this way in reality.

The lipid core size was defined as a ratio in percentage of the lipid core volume to the total plaque volume. All the 13 cases cover the changes of lipid core size from 24.46% to 12.23%, and the fibrous cap thickness from 0.15 to 0.64 mm. For the largest lipid core case, only one case with the thinnest fibrous cap can be generated (Case 1), because a thicker cap with a larger lipid core will push the lipid core very close to the arterial outer wall causing simulation difficulties.

2.2. Material properties and boundary conditions

The carotid arterial wall was assumed to be nonlinear, isotropic, and incompressible. The five-parameters Monney–Rivilin model was used to describe the material properties of the arterial wall. The strain energy function is given by

$$W = C_{10}(I_1 - 3) + C_{01}(I_2 - 3) + C_{20}(I_1 - 3)^2 + C_{11}(I_1 - 3)(I_2 - 3) + C_{02}(I_2 - 3)^2 + \frac{1}{d}(J - 1)^2 \quad (1)$$

where I_1 and I_2 are the first and second strain invariants, d is the material incompressible parameter, J stands for the ratio of the deformed volume over the undeformed volume of materials, C_{10}, C_{01}, C_{20}, C_{11} and C_{02} are material constants which need to be decided by the experimental data. In the study, the following values were chosen to match the existing literature (Tang et al., 2004b) using curve-fitting method in Ansys: $C_{10} = 50.445$ kPa, $C_{01} = 30.491$ kPa, $C_{20} = 40$ kPa, $C_{11} = 120$ kPa, $C_{02} = 10$ kPa, and $d = 1.44 \times 10^{-7}$ Pa^{-1}. The stiffness of lipid core is about 1/100 of fibrous cap or less. Since the variation of lipid core stiffness only has minor impact on the simulation results (Tang et al., 2004a), the lipid core was assumed to be very soft with 2 kPa Young's modulus and 0.49 for Poisson's ratio which are similar to the values used in Finet et al. (2004). The interface between lipid core and arterial wall was treated as a so-called "always bonded" contact; the pure penalty method was employed for the contact connection. Computational nodes at the inlet plane of common carotid artery (CCA) were fixed in all

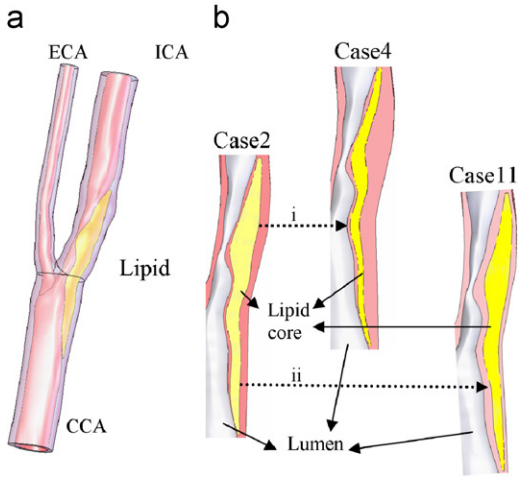

Fig. 1. Diagrams of arterial model reconstruction and manipulation of simulation cases. (a) The reconstructed geometry for Case 2 (base model). (b) Schematic reconstruction of simulation cases: (i) fibrous cap thickness remains constant and lipid core volume is reduced proportionally, from Case 2 to 4; (ii) lipid core volume remains constant, and the fibrous cap thickness is increased proportionally, from Case 2 to 11. CCA: common carotid artery, ECA: external carotid artery, ICA: internal carotid artery.

Table 1
Descriptions of the plaque geometry of the simulation cases constructed in the study

Fibrous cap thickness (mm)	Lipid core size			
	26.46%	22.4%	18.15%	12.23%
0.15	Case 1	Case 2	Case 3	Case 4
0.35		Case 5	Case 6	Case 7
0.58		Case 8	Case 9	Case 10
0.64		Case 11	Case 12	Case 13

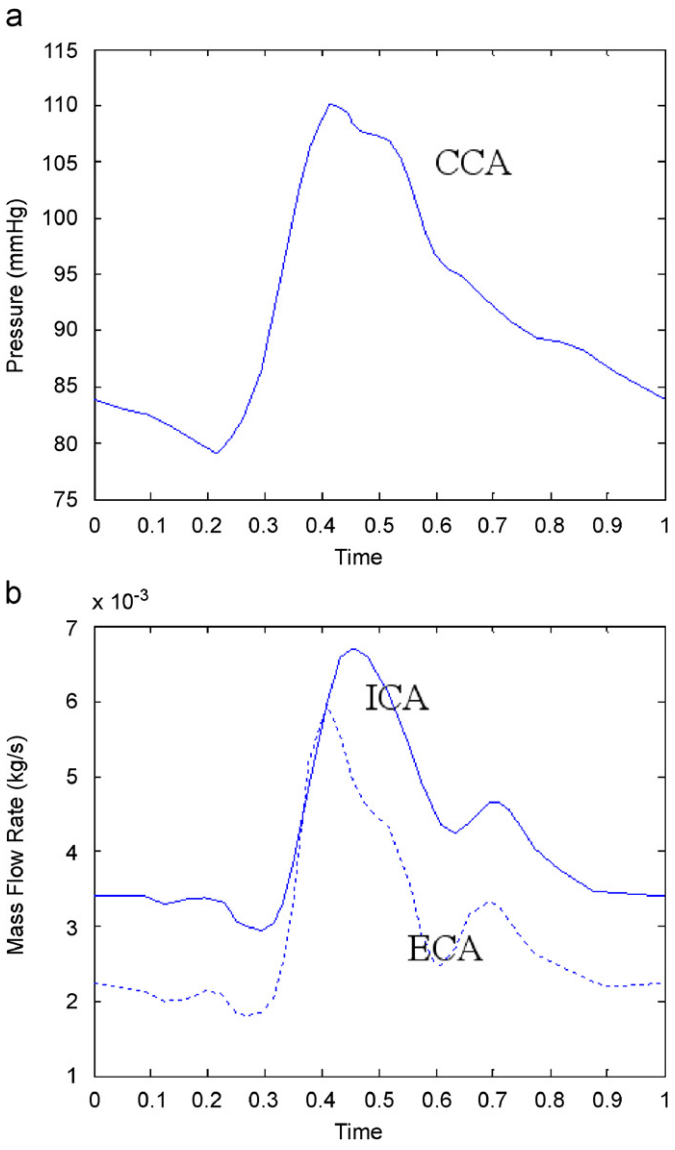

Fig. 2. Fluid boundary conditions for the inlet and outlet planes. Time is normalized by the cardiac period in x-axis: (a) pressure for CCA and (b) mass flow rates for ICA and ECA, respectively.