PLAQUE STRUCTURE AFFECTS MECHANICAL STRESS DISTRIBUTION WITHIN BLOOD VESSELS

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ABSTRACT
The main goal of this study is to investigate the effects of plaque structure on its stress distribution. Rupture of plaque causes cerebrovascular diseases which lead to high mortality rates all over the world. Computers are powerful tools to understand the mechanism of plaque rupture. In this study, 3D fluid structure interaction simulation is constructed in ABAQUS 6.13 to clarify the relation between stress distribution of plaque and its structure. A model of common carotid artery with distributed stenosis was chosen for the simulation. To investigate the effects of plaque structure on stress distribution, thickness of fibrous cap and lipid core size were varied in the stenosis. Furthermore material properties of plaque were changed to study their effects on stress distribution. The models were simulated under pulsatile flow during the heart cycle and the stress distribution, maximum plaque wall stress and the flow shear rates were analyzed. The results of this study show that the viscoelastic properties of the plaque and the vessel wall reduces flow shear rate. Moreover it was shown that the thickness of fibrous cap is a more important parameter than lipid core size and the maximum plaque wall stress is more dependent to fibrous cap thickness rather than the lipid core size.

KEY WORDS
Plaque rupture, Common carotid artery, Atherosclerotic plaque, Fluid structure interaction, Viscoelastic properties.

1. Introduction

The rupture of atherosclerotic plaque is the main cause of cardiac syndromes such as stroke and heart attack that leads to extensive mortality in the world [1, 2]. In the past decade there have been wide investigations associated with the medical imaging and fluid solid interaction (FSI) simulation to discover what causes the plaque rupture. Despite many researchers have investigated this phenomenon, its mechanism has not been understood completely. The researches have shown that mechanical parameters such as flow shear stress, flow shear rate and plaque wall stress have great influences on developing plaque rupture [3-6], but the exact effects of these parameters on plaque rupture and the critical quantities of these parameters that start plaque rupture have not been realized clearly. There are some researches that have reported some of the critical values of these mechanical parameters for starting plaque rupture [7-9]. Teng and coworkers investigated the effects of the mechanical parameters on six samples and quantified the critical plaque wall stress and flow shear stress and reported which parameter is a better predictor for the site of rupture [9]. Hung and coworkers also used a MRI based approach and FSI simulation to quantify the critical shear stress and plaque wall stress of 12 patients [8].
The accurate stress/strain distribution in a plaque is vital to calculate the critical values of the mechanical parameters in developing plaque rupture. Stress/strain distribution in a plaque is strongly dependent on plaque structure including lipid core size, fibrous cap thickness, stenosis severity and its material properties [10, 11], so it is important to know how plaque structure influences the mechanical parameters which causes developing plaque rupture. This research tries to find the role of plaque structure on these mechanical parameters during a heart cycle with pulsatile flow. In this study, FSI simulation of common carotid artery with distributed plaque was

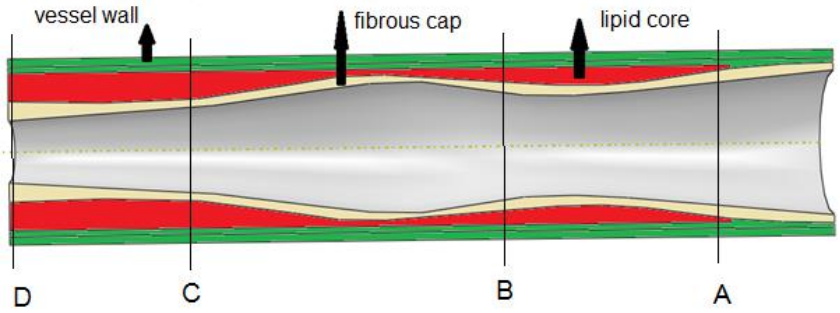

Figure 1: Common carotid artery with atherosclerotic plaques

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performed and stress distribution, flow shear rates, maximum plaque wall stress and its critical sites were analyzed. In the first model, plaque structure had a composite form with lipid core and fibrous cap, in the second model plaque had unit structure without lipid core. Sizes of both fibrous cap and lipid core have been varied in the stenosis to understand their influences on the results. To find out the effects of viscoelastic properties of plaque and vessel wall on stress/strain distribution, the composite structure was modeled with and without viscoelastic properties. Viscoelastic properties of these tissues were not considered in the previous studies.

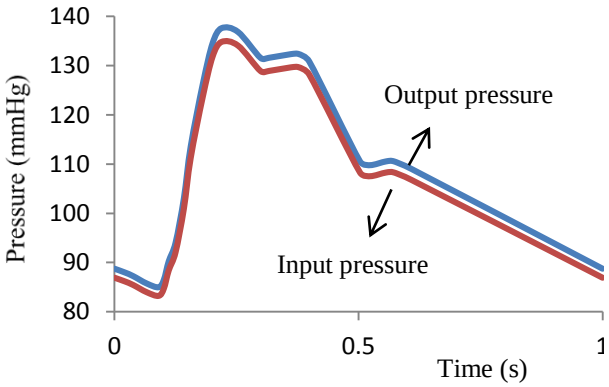


Figure 2: Pressure profile of inlet and outlet during heart cycle

2. Material and method

FSI simulation of common carotid artery with distributed stenosis was performed in ABAQUS. No-slip interaction and continuity of displacement were considered between solid and fluid interface. Flow was assumed as laminar, incompressible with a viscosity of 3.8×10^{-3} Pa s and a density of $1050\ \mathrm{kg\ m^{-3}}$. Diameter, thickness and length of common carotid artery were assumed to be 7.5 [12], 3.8 and 40 mm respectively. As shown in figure 1, the geometry of stenosis was modeled with various thicknesses of fibrous cap and lipid core size to investigate the effects of fibrous cap thickness and lipid core size on the stress distribution.

Time dependent pressure profiles at inlet and outlet of common carotid artery were imposed (figure 2). The profiles were extracted from the literature in which systolic and diastolic pressure of patients were considered as the maximum and minimum pulsatile pressure waveform [13]. The outlet and inlet were fixed in both radial and axial directions.

Material properties of plaques were extracted from literature. Heinland and coworkers considered plaques as both composite and unit structures and derived their constitutive parameters [2]. In the unit structure, plaque was considered as a neo-Hookean material (equation 1). In the composite structure, for fibrous cap and arterial wall tissues, GOH[1] model (equation 2) was used to describe tissues' anisotropic hyperplastic properties and for the lipid core, neo Hookean model was used [2]. Constant parameters of the constitutive models are presented at table 1.

Neo Hookean model: $W = E(I_1 - 3)$ (1)

GOH model [14]:

$$W = C_{10}(I_1 - 3) + \frac{k_1}{2k_2}\exp\left(k_2\left(\frac{I_3}{3} - 1\right)^2 - 1\right) \quad (2)$$

3. Result

In the first model, structural of plaque was considered as composite material with lipid core and fibrous cap and in the second model it was considered as unit material.

3.1 Composite structure of plaque with viscoelastic properties

Figure 3 shows the maximum principal stress of plaque

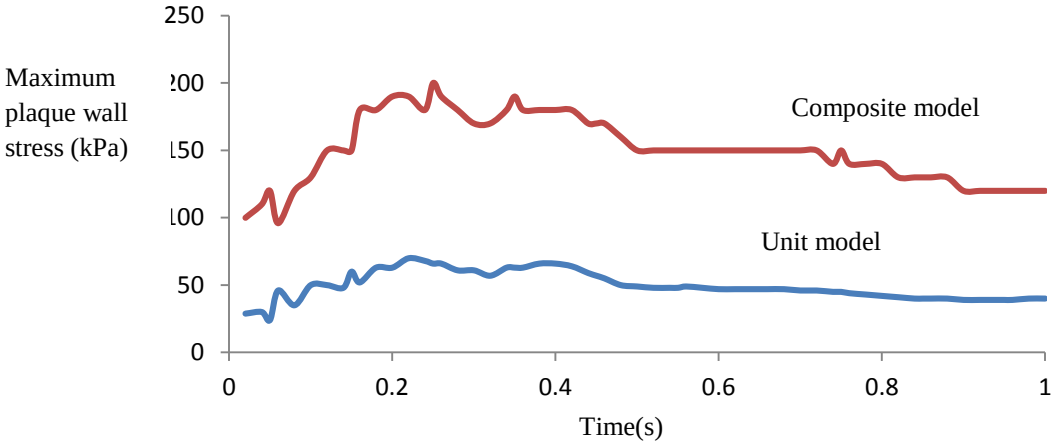


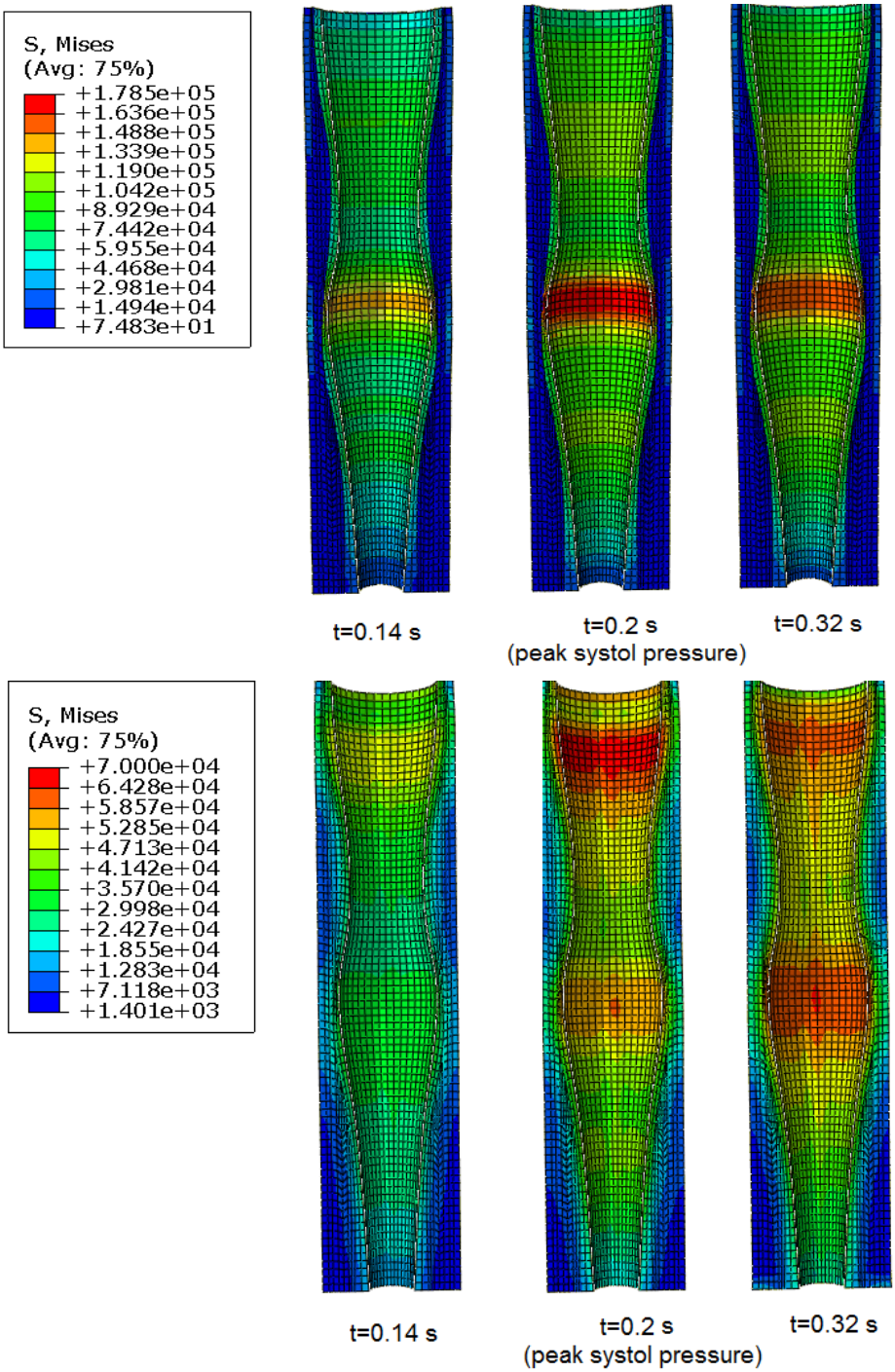
Figure 3: Comparison maximum principal plaque wall stress between composite and unit models

[1] Abbreviation of Gasser-Ogden-Holzapfel model

wall at 20 steps during heart cycle for both models. Maximum principal plaque wall stress of composite

Table 1: Constitutive parameters of material properties

Plaque structure	C_{10}(MPa)	k_1(MPa)	k_2	g_1	g_2	g_3	g_4	g_5
composite	0.056	41.08	1749	0.1595	0.1177	0.0623	0.1612	0.2101
unit	0.0096	0.324	41.14	0.1112	0.1606	0.1328	0.1423	0.107



model is 200kPa which is at the range of plaque wall

Figure 4: Maximum principal stress counter (Pa) at 3 steps, during heart cycle. (a) Composite structure of plaque. (b) Unit structure of plaque

Table 2: Comparison of shear rates of composite structure of plaque with and without viscoelastic properties

time	0.08	0.18	0.20	0.22
Model with viscoelastic property	2.56e3	3.28e3	3.0e3	4.10e3
Model without viscoelastic property	5.60e3	5.38e3	4.38e3	5.08e3
difference	119%	64%	46%	24%

stress of common carotid artery in the literature and is compatible with the previous experimental studies that have been done on patient specific [6, 9].
Figure 4 shows Stress distribution of the both models in 3 steps during heart cycle. In the composite model the zone of maximum plaque wall stress always happens at the thinner fibrous cap. The maximum plaque wall stress in this site is 200% higher than other zones of rupture. These observation is also compatible with the previous experimental studies [11, 15]. Lipid core and vessel wall have lower wall stress than fibrous cap; lipid core has also a negative maximum principal stress. This shows that lipid core is always under pressure during heart cycle.
Furthermore at section C-D of figure 1, it can be seen that while thickness of fibrous cap was increased and the size of lipid core was not change, wall stress of fibrous cap decreases. At the section A-B, it can be seen that while the lipid core size was increased and the thickness of fibrous cap was not changed, there is no considerable change in the wall stress of plaque. These show that the plaque wall stress is more dependent on the thickness of the fibrous cap than lipid core size. Moreover a thinner fibrous cap leads to an increase in maximum wall stress.
In the both A-B and C-D sections there are not considerable changes for wall stress of lipid core. It means that varying fibrous cap thickness and lipid core size does not change lipid core stress considerably.

3.2 Unit structure of plaque with viscoelastic properties

As shown in figure 3 maximum plaque wall stress of unit model is 70 kPa. Thinner fibrous cap and the zone near the input are Critical sites of plaque wall stress. In this model lower stress is observed at outer layer. At section C-D increasing thickness of plaque reduces plaque wall stress.

3.3 Composite structure of plaque with and without viscoelastic property

As can be seen in table 2, there is a comprehensive difference between the shear rates of the composite model with and without viscoelastic properties. Shear rates have been shown at four steps. These steps are near the peak of the systole pressure.

4. Discussion

Previous studies have shown that the mechanical properties of plaque can affect stress distribution [10, 11]. In this study FSI model of common carotid artery with plaque atherosclerotic was developed to investigate the effects of mechanical properties and structure of plaque on stress distribution, maximum plaque wall stress, its critical sites and flow shear rates. It was shown that the stress distributions of the two models have considerable differences. At the peak systole pressure (t=0.2 s), the maximum plaque wall stress of the first and the second model are about 190 kPa and 70 kPa respectively. The difference between the two models is more than 200% at the peak systole pressure. At the other steps, major differences between maximum principal stresses are observed too. The average of maximum plaque wall stress in the first model is about 150 kPa which is 300% higher than the average of maximum plaque wall stress in the second model. The main reason for the considerable difference between the values of principal wall stress in the two models is sprung from lipid core elimination. Since lipid core has softer structure than fibrous cap and vessel wall, elimination of lipid core causes an unrealistic increase in the whole structure strength. This leads to a decrease in deformation and a reduction in the stress value of the plaque wall.
Critical zone at the composite model happens at thinner fibrous cap which is compatible with experimental studies [11, 15]. Unit model predicts critical zone at both thinner fibrous cap and the zone near the input, (Figure 4). As a result composite model predicts critical zone more accurately.
Both models predict lower wall stress at the outer layer, this observation has also reported in the other studies on patient [16]. Furthermore in the composite model, lipid core is under pressure while in the unit model it is under tension. So if lipid core is neglected in the model, stress distribution of this zone becomes completely different.
Varying lipid core size at section A-B causes no change in

the stress distribution of fibrous cap while increasing fibrous cap thickness at section C-D leads to reduction in its stress distribution. It seems that fibrous cap thickness has more effects on stress distribution than lipid core size. Furthermore in both sections stress distribution of lipid core does not change considerably. As a result stress distribution of lipid core is not affected by its size and fibrous cap thickness.

Viscoelastic properties of plaque changes shear rates of flow which has a major role in developing plaque rupture [3]. According to table 2, elimination of viscoelastic properties of plaque and vessel wall can increase shear rates of the models up to 119%.

5. Conclusion

In this study, it was shown that the maximum plaque wall stress, its critical sites and flow shear rates of blood are dependent to the structure of plaque and its mechanical properties. Since elimination of lipid core at plaque structure causes a reduction in the maximum plaque wall stress up to 300%, it is important to model plaque as a composite material including both fibrous cap and lipid core. However, at the composite structure of plaque, the thickness of the fibrous cap is a more important parameter than the lipid core size in order to determine the maximum plaque wall stress. Viscoelastic properties of plaque and vessel wall reduce flow shear rates up to 120%; so for calculating critical values of flow shear rates, it is important to consider viscoelastic properties.

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