# Effects of lipid core stiffness and cap thickness on atherosclerotic plaque wall stress

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**Abstract :** Rupture of atherosclerotic plaque is related to mechanical stress, plaque geometry and the material properties of plaque tissues. An ideal three dimensional coronary artery with eccentric stenosis was modelled, and stress analysis with fluid structure interactions was performed using ADINA in order to investigate the effects of cap thickness and lipid core stiffness on atherosclerotic wall stress under pulsatile flow in coronary artery. Three different cap thickness (45, 65 and 200) and core stiffness (soft, intermediate and hard) were considered. Lipid core stiffness effect on stress and strain increased as the cap thickness decreased. The maximum peak cap stress increased up 250%, 225% and 152%, respectively as core stiffness decreased. Therefore, a very thin cap with soft lipid core may increase the vulnerability of atherosclerotic coronary plaque.

## 1. Introduction

Coronary artery disease is one of the major causes of death throughout the world. The rupture of the atherosclerotic plaque is related to the mechanical stress and structural integrity of plaque wall tissues (1). The vulnerable plaque ruptures when the mechanical stress in the cap tissue exceeds the ultimate strength of it. The peak cap stress is determined by arterial pressure loading, plaque wall morphology, and the mechanical property of plaque tissue. Computational stress analysis has been performed in order to estimate the plaque stress using ideal plaque model (2). The effects of cap thickness have been investigated extensively, and the numerous computational studies have shown that the stresses in the cap increase exponentially with decreasing the cap thickness (3). However, the correlation of core stiffness and cap stress has not been considered. In this study, an idealized three dimensional stenotic plaque model which had a thicker downstream cap tissue were constructed.

## 2. Method

Three-dimensional coronary artery models with eccentric stenosis were modeled and wall stress was analyzed considering fluid and structure interaction (FSI). The diameter of the lumen was 3 mm and the vessel wall thickness was 1 mm. The length of the stenosis was 15 mm and the luminal height of throat was 1.7mm. The stenotic wall consisted of the lipid core and fibrous cap, and models with three different cap thicknesses (200, 65, and 45  $\mu$ m) were analyzed. The 4-node tetrahedral elements were used for the fluid domain, and the 8-node hexahedral elements were used for the wall domain. Finer meshes were used in the plaque cap and the lipid core zone to accommodate abrupt changes in geometry.

Distally thickened model represented the atherosclerotic stenosis with pronounce distal cap thickening found in the clinical observation. Different mechanical properties were applied to lipid core in order to clarify the effect of the core stiffness on wall mechanics.

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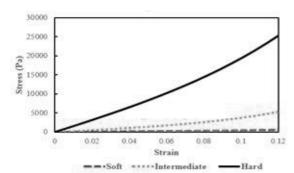


Fig. 1 Stress-strain curves for three different core tissues

For models, the fluid was assumed to be incompressible and Newtonian, and the density and viscosity of the fluid was 1,050 kg/m³and0.0035kg/m.·s, respectively. No slip boundary condition was used on the wall. The Navier-Stroke equation with arbitrary Lagragian-Eulerian formulation was used to solve the governing equation in the presence of fluid structure interaction (FSI), and transient implicit scheme was applied to flow. The material properties of the arterial wall and plaque tissues were assumed to be hyperelastic, isotropic, and incompressible. Three mechanical lipid cores with different stiffness (soft, intermediate, and hard) were applied in the lipid core in order to study the effects of core stiffness.

# 3. Result and discussion

The maximum peak stress and strain were found at the proximal site of stenosis. The maximum peak cap stress increase as cap thickness decrease, and the maximum value in the 45 µm thick soft lipid core was 354.59 kPa. The maximum peak cap stress increased more than 320% in the 45 µm thick model with soft lipid core comparing to the 200 µm thick model (Table. 1). Core stiffness effect on the maximum peak cap stress was relatively less in the 200 µm thick model. However, the maximum peak cap stress increase up 250% 225%and 152% in the 45µm, 65µm and 200µm thick models, respectively, when core stiffness changed from hard to soft.

Table1. Cap stress at peak pressure

| Core         | 45µm   | 65µm   | 200µm  |
|--------------|--------|--------|--------|
| Soft         | 354.59 | 221.8  | 110.17 |
| Intermediate | 293.13 | 191.67 | 103.13 |
| Hard         | 141.11 | 99.44  | 72.72  |

This result implied that the maximum peak cap stress increased due to core stiffness (soft core leads high stress) in the thin.

## 4. Conclusion

The effect of cap thickness and core stiffness on cap stress and strain was prominent. The core stiffness increased the cap stress more noticeably as the cap thickness decreased less than 65µm. Therefore, the plaque with the thinner cap and softer lipid core may increase the vulnerability of atherosclerotic coronary plaque.

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