IDENTIFYING VULNERABLE PLAQUES USING MRI-BASED FINITE ELEMENT ANALYSIS

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INTRODUCTION

Atherosclerotic vascular disease is the most common cause of morbidity and mortality in the world. Plaque rupture has been identified as a critical step in the evolution of arterial plaques[1], whereas the biomechanics of plaque rupture is still not fully understood[2,3]. To explore the role of biomechanics in the assessment of risk of rupture in carotid atheromatous plaque using high-resolution MRI, we used finite element analysis based on the magnetic resource imaging (MRI) method to simulate the mechanism of carotid plaque in asymptomatic and symptomatic subjects.

METHODS

Sixteen subjects, eight symptomatic and eight asymptomatic, underwent high-resolution multi-sequence in vivo MR imaging of the carotid bifurcation. All the imaging studies were conducted on a 1.5 Tesla whole body system using a customized four-channel phased array coil wrapped around the neck. The following axial 2D, ECG-gated, blood-suppressed, fast spin echo pulse sequences were used: intermediate T2 weighted (TR/TE: 2*RR/46) with and without fat saturation, T2 weighted (TR/TE: 2*RR/100), STIR (TR/TE/TI: 2*RR/46/150), and fat-suppressed T1 weighted (TR/TE: 1*RR/ 7.8). The pixel size was 0.4x0.4x3mm in all cases. Segmentation was based primarily on the STIR sequence, which was deemed to be most accurate for fibrous cap and lipid core, when compared with histology. Each patient axial MR slices were segmented manually and all contours were traced to generate the boundaries of lipid core, fibrous cap, vessel lumen and wall based on net intensity characteristics. The mesh was generated for each slice and finite element analysis was conducted in each case to determine the stresses within every plaque. The linear elastic model was used to model the plaque components. The material properties (Young's modulus and Poisson's ratio) were chosen based on recent publications[4]. In this study, the Young's moduli used were: arterial wall 16.8 kPa, fibrous cap 700 kPa and lipid core 6.25 kPa, and a Poisson's ratio of 0.49999 was used for all components. The internal luminal pressure was assigned at 15kPa. Statistical analysis was carried out for the comparison of symptomatic and asymptomatic patients.

RESULTS AND DISCUSSION

Analysis was carried out for every slice that covers the plaque along the vessel for each patient. The peak stresses are illustrated to be within the disease part of the vessel. The slice with peak stress was chosen for each patient for the comparison between symptomatic and asymptomatic patients. High stress concentration happened at the shoulder region of the symptomatic patient and the maximum stress is 344.2 kPa, while in asymptomatic patient, the stress distribution is much more averaged and the maximum stress is 150 kPa. The mean Principal stresses in the plaques of symptomatic patients are significantly higher than those of asymptomatic patients (533.4±185.3 versus 203.5±121.3 kPa, $p<0.1$). In Figure 1, mean stress with standard deviation is shown for symptomatic and asymptomatic patients and this demonstrates a significant difference between these two groups.

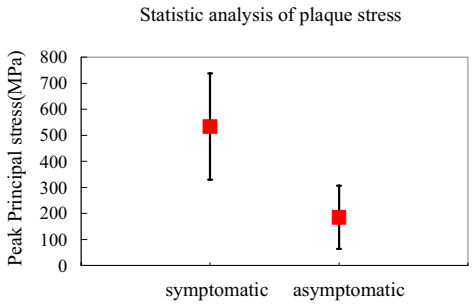

Figure 1: Comparison of plaque mean stress with standard derivation between symptomatic and asymptomatic patients

CONCLUSIONS

This Finite element analysis suggests that the maximum stresses within the plaques of symptomatic patients are higher than those of asymptomatic patients. Mechanical stress in the carotid plaque, therefore, ought to be regarded as a complementary indicator of plaque rupture risk alongside the traditional measure of stenosis. A combination of high resolution MR and finite element analysis could potentially act as a useful tool for assessment of risk in patients with carotid disease.

REFERENCES

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