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Study of carotid arterial plaque stress for symptomatic and asymptomatic patients

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ABSTRACT

Stroke is one of the leading causes of death in the world, resulting mostly from the sudden ruptures of atherosclerosis carotid plaques. Until now, the exact plaque rupture mechanism has not been fully understood, and also the plaque rupture risk stratification. The advanced multi-spectral magnetic resonance imaging (MRI) has allowed the plaque components to be visualized *in-vivo* and reconstructed by computational modeling. In the study, plaque stress analysis using fully coupled fluid structure interaction was applied to 20 patients (12 symptomatic and 8 asymptomatic) reconstructed from *in-vivo* MRI, followed by a detailed biomechanics analysis, and morphological feature study. The locally extreme stress conditions can be found in the fibrous cap region, 85% at the plaque shoulder based on the present study cases. Local maximum stress values predicted in the plaque region were found to be significantly higher in symptomatic patients than that in asymptomatic patients (200 ± 43 kPa vs. 127 ± 37 kPa, $p=0.001$). Plaque stress level, defined by excluding 5% highest stress nodes in the fibrous cap region based on the accumulative histogram of stress experienced on the computational nodes in the fibrous cap, was also significantly higher in symptomatic patients than that in asymptomatic patients (154 ± 32 kPa vs. 111 ± 23 kPa, $p<0.05$). Although there was no significant difference in lipid core size between the two patient groups, symptomatic group normally had a larger lipid core and a significantly thinner fibrous cap based on the reconstructed plaques using 3D interpolation from stacks of 2D contours. Plaques with a higher stenosis were more likely to have extreme stress conditions upstream of plaque throat. The combined analyses of plaque MR image and plaque stress will advance our understanding of plaque rupture, and provide a useful tool on assessing plaque rupture risk.

1. Introduction

Atherosclerosis is one of the leading causes of death all over the world, caused by plaque rupture and subsequent thrombus formation (Rosamond et al., 2007). Despite many years of research, the underlying mechanism for plaque rupture is still not fully understood (Naghavi et al., 2003). It is believed that several factors play important roles in the rupture process: (a) biological abnormalities, such as enhanced inflammatory activity, and the accumulation of macrophages (Libby et al., 2002); (b) biomechanical factors, such as local extreme mechanical stress (Richardson et al., 1989; Cheng et al., 1993; Lee et al., 1993; Ohayon et al., 2001). With recent development of imaging techniques and the improved understanding of plaque rupture mechanisms, it is increasingly becoming clear that plaque vulnerability cannot be described by the degree of stenosis alone (Giroud et al., 1992). It has been suggested that plaque morphology, plaque biomechanical environment, and inflammation activity will all influence their vulnerability (Richardson, 2002; Naghavi et al., 2003; Ohayon et al., 2008). From biomechanical aspect, the rupture can be considered to be a mechanical failure event. Therefore it is useful to study the detailed mechanical stress distribution on specific plaques in order to develop a more precise assessment of the risk of plaque rupture (Zheng et al., 2005; Li et al., 2006).

Patient specific plaque stress analysis has been providing critical information on the understanding of plaque rupture mechanisms and may eventually lead to rupture risk assessment (Tang et al., 2005a). Recent developments in high-resolution multi-spectral MRI have allowed plaque components to be

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