ACUTE MECHANICAL RESPONSE OF HUMAN CORONARY FIBROATHEROMAS TO STENTING

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INTRODUCTION

According to estimates, approximately 1.1 million Americans suffered an acute myocardial infarction (AMI) or heart attack annually. Over 45% of the individuals who suffer an AMI within a given year are expected to die as result of the trauma (2002 Heart and Stroke Statistical Update, American Heart Association).

Over the past several years the research community and physicians have altered the historical belief that an AMI is caused by a total arterial occlusion that results from the slow progressive narrowing of a coronary artery. In this scenario, the progression of the disease is believed to result in the development of stable atherosclerotic lesions. However, the paradigm that is now accepted assigns fault of an AMI to an acute closure of the artery that occurs in response to the rupture of a fibrous cap that overlays an accumulation of lipid and other materials with low structural strength within the vessel wall. Upon rupture, the thrombogenic contents of the lipid core come into contact with the blood and, in turn, initiate a clotting response. If the body is unable to minimize the build up of thrombus, the clot can progress and yield a total occlusion of the coronary artery within a short period of time. These unstable atherosclerotic lesions that are prone to rupture are distinguished from stable lesions by the presence of sub-intimal lipid accumulations with a thin fibrous cap isolating the thrombogenic material from the blood. This class of lesions has begun to be identified as thin-capped fibroatheromas (TCFAs) [1].

PTCA alone or with stent implantation has become the physician's treatment of choice for occlusive atherosclerotic lesions. The American Heart Association estimated that in 1999 over one million interventional procedures were performed in the United States. With the overwhelming success of these therapies, the number of interventional procedures that are performed per year has since grown.

A significant amount of research by both the academic and medical communities has gone into improving the outcome of PTCA and stenting. Much of this research has gone into understanding the mechanical interactions between the tissue and the interventional device (angioplasty balloon or stent) and the mechanical impact of device delivery [2]. As our understanding of TCFAs grows, the need to understand how current interventional technology would interact with these unstable lesions becomes increasingly important.

In 1989, Richardson, et al. [3] correlated the location of fibrous cap rupture in patients who had died of coronary thrombosis with stress concentrations seen in idealized finite element models of simulated arteries with fibroatheromas. The histological research showed that 42 (63%) of 67 eccentric plaques with lipid accumulations ruptured within the shoulder region. The location of rupture corresponded to the location of stress concentrations as predicted by mechanical models with eccentric plaques and lipid accumulations. Many investigators have since applied finite element techniques to predict biomechanical behavior of de novo atherosclerotic lesions in response to in vivo loads.

In this ongoing study, the authors are investigating the acute mechanical response of coronary fibroatheroma plaques to stenting. This paper presents the general mechanical modeling methodology as well as results from the initial data relating the impact of stenting on stresses within the fibrous cap.

METHODS

The current study utilizes geometry data gathered from a Guidant sponsored clinical study in which a novel intravascular optical coherence tomography (OCT) system (Wellman Laboratories of Photomedicine & Massachusetts General Hospital, Boston, MA) is used to image flow-limiting fibroatheromas in live patients undergoing a standard PTCA procedure. A data set is selected for subsequent mechanical modeling based on a primary factor and a collection of secondary factors. The primary criterion for model development of the fibroatheroma is the existence of significant sub-intimal lipid accumulation identified in the OCT images [4]. However, other morphological features such as number of lipid accumulations present

within a lesion, lumen contour, lesion eccentricity and fibrous cap thickness contribute as secondary criteria.

The fibroatheromas of interest are subjected to a simple, semi-automated tracing technique in order to segment the primary morphological features. This process defines the lumen boundary, the external elastic lamina, the fibrous cap thickness and the shape of any lipid accumulations. Figures 1A and 1B illustrate two of the fibroatheromas selected for mechanical modeling.

The three constituents composing the fibroatheroma cross-sections are each modeled as isotropic, hyperelastic materials. The material data used to characterize the stress-strain behavior employed in the current study are derived from two sources. Work by Beattie, et al. [5] provides input data for material characterization of all three constituents gathered from human aortic specimens. An independent study by Feezor, et al. [2] provides a secondary set of data characterizing the mechanical behavior of medial tissue from experiments performed on human coronary specimens. The technique used to develop the final hyperelastic material models from data for each constituent has been presented previously. [2]

With lesion features identified and traced, the segmented vessel is imported into a finite element preprocessor for discretization and integration with a 2-dimensional stent model. Seven different stent cross-sections are utilized in this study. Each cross-section possesses strut characteristics (width, thickness, distribution, etc.) that are consistent with the class of coronary stents currently available to physicians. Loading of the fibroatheroma consists of a contact load defined between the lumen surface of the fibroatheroma and each of the stent struts. In turn, each of the struts is assigned an appropriate displacement so that the final lumen diameter is 3.0 mm.

RESULTS

Figures 2A and 2B show the impact of stenting on a fibroatheroma when subjected to the expansion of a 2-dimensional stent pattern to a final lumen diameter of 3.0 mm. Figure 3 indicates one metric that is extracted from these models. It illustrates the maximum principal stress in the plaque shoulder region as predicted by the computational models of each fibroatheroma presented in Figures 1A and 1B averaged over all seven stent patterns. A statistically significant difference in maximum principal stress within the shoulder region of 120% exists between the two fibroatheroma models of differing histological composition ($P < 0.01$).

DISCUSSION

The clinically based computational models presented in the current study show that stress concentrations arise in shoulder regions of the fibrous plaque cap of coronary fibroatheromas during stent deployment. These results agree with the histological study and mechanical models of idealized de novo plaques originally presented by Richardson, et al. [3]. The significant difference in the magnitude of these stress concentrations indicates that lesion composition and morphology can dramatically impact the loads placed on the arterial tissue during the implantation of an interventional device. The knowledge gained from the present and future studies continues to influence the scientific evaluation of techniques and devices applied in the treatment of fibroatheromas and other atherosclerotic lesions.

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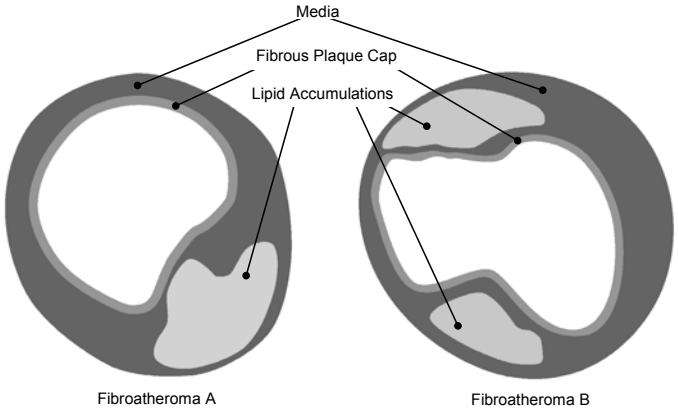
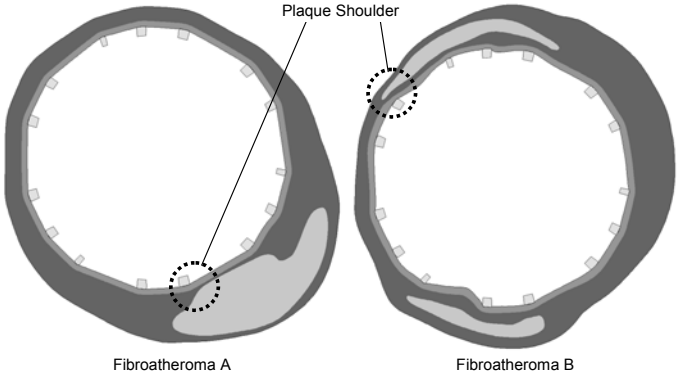


Figures 1A & 1B: Sample fibroatheromas



Figures 2A & 2B: Sample fibroatheromas post expansion with characteristic stent profile

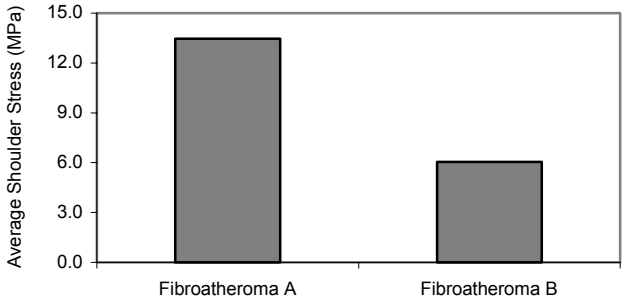


Figure 3: Comparison of the maximum principal stress in the plaque shoulder of each fibroatheroma