Journal of Biomechanics 47 (2014) 878–889

Contents lists available at ScienceDirect

Journal of Biomechanics

journal homepage: www.elsevier.com/locate/jbiomech
www.JBiomech.com

Numerical simulation of arterial dissection during balloon angioplasty of atherosclerotic coronary arteries

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ARTICLE INFO

Article history:
Accepted 13 January 2014

Keywords:
Balloon angioplasty
Coronary artery
Arterial dissection
Cohesive zone
Finite element model
Plaque fracture

ABSTRACT

Balloon angioplasty is a standard clinical treatment for symptomatic coronary artery disease. In this procedure, controlled damage is applied intraluminally to the wall of a stenotic artery. Dissection of the coronary artery is a commonly observed clinical complication of angioplasty; however, not all dissections can be detected angioscopically. This work focuses on studying the dissection mechanisms triggered during the early stages of angioplasty in an atherosclerotic coronary artery, addressing the problem by means of a parametric study based on a simplified finite element model and cohesive interface modeling. Our results emphasize the presence of several damage mechanisms, at different locations, that are triggered near the very beginning of the process and evolve competitively, depending on both geometry and material properties of the atherosclerotic vessel. Small-scale damage was evidenced, which would not be detectable by angiography or intravascular ultrasound, but could potentially be sufficient to stimulate smooth muscle cell activation, promoting late-onset complications such as restenosis.

1. Introduction

In advanced atherosclerosis, plaque development can lead to flow-limiting stenosis, resulting in myocardial ischemia accompanied by symptoms such as stable angina. Balloon angioplasty is a standard clinical treatment for symptomatic coronary artery disease. In this procedure, controlled damage is applied intraluminally to the wall of a stenotic artery in order to restore blood flow and to prevent ischemia of the downstream tissue. This induced damaged can take several forms, including irreversible deformation of the tissue caused by overstretch during balloon inflation, plaque fracture, and dissection of the plaque from the underlying vessel wall (Fitzgerald et al., 1992; Honye et al., 1992; Tenaglia, 1997). Since arterial wall damage is thought to contribute over the long term to restenosis, leading to eventual re-occlusion of the treated vessel, considerable effort has gone into understanding the mechanisms and consequences of each mode of damage. Computational simulations of arterial damage due to overstretch and inelastic deformation (Balzani et al., 2012; Gasser and Holzapfel, 2007a) or to plaque fracture (Ferrara and Pandolfi, 2008; Gasser and Holzapfel, 2007b) have been reported in the literature.

Dissection of the coronary artery is a commonly observed clinical complication of angioplasty (Honye et al., 1992). Coronary artery dissection can lead to myocardial infarction if the dissected layer partially or totally occludes the vessel lumen, leading to downstream ischemia. Substantial dissections appear as angioscopic filling abnormalities or as overt separation of layers when viewed by intravascular ultrasound (IVUS) (Honye et al., 1992). These may be treated by additional stenting at the time of the original intervention. However, not all dissections can be detected angioscopically. Another type of damage noted clinically is intramural hematoma, or accumulation of blood within the arterial wall, which is most readily identified by IVUS or by intravascular optical coherence tomography (OCT). In one clinical series, a large percentage (86%) of intramural hematomas could be traced to propagation of an intimal tear; however, a smaller number demonstrated no apparent connection with the lumen by IVUS (Maehara et al., 2002), suggesting that other mechanisms such as damage to the vasa vasorum and subsequent hemorrhage may contribute to their development (Nalbandian and Chason, 1965). Arterial dissection or intramural hematoma which is not detected at the time of the original angioplasty can lead to emergent re-occlusion of the treated vessel (Maehara et al., 2002; Tenaglia, 1997).

Based on recent experimental work that demonstrated a considerable range of variation in tearing strength of human left anterior descending (LAD) coronary arteries (Wang et al., 2013b), we investigated the question of whether some patients may be

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http://dx.doi.org/10.1016/j.jbiomech.2014.01.009

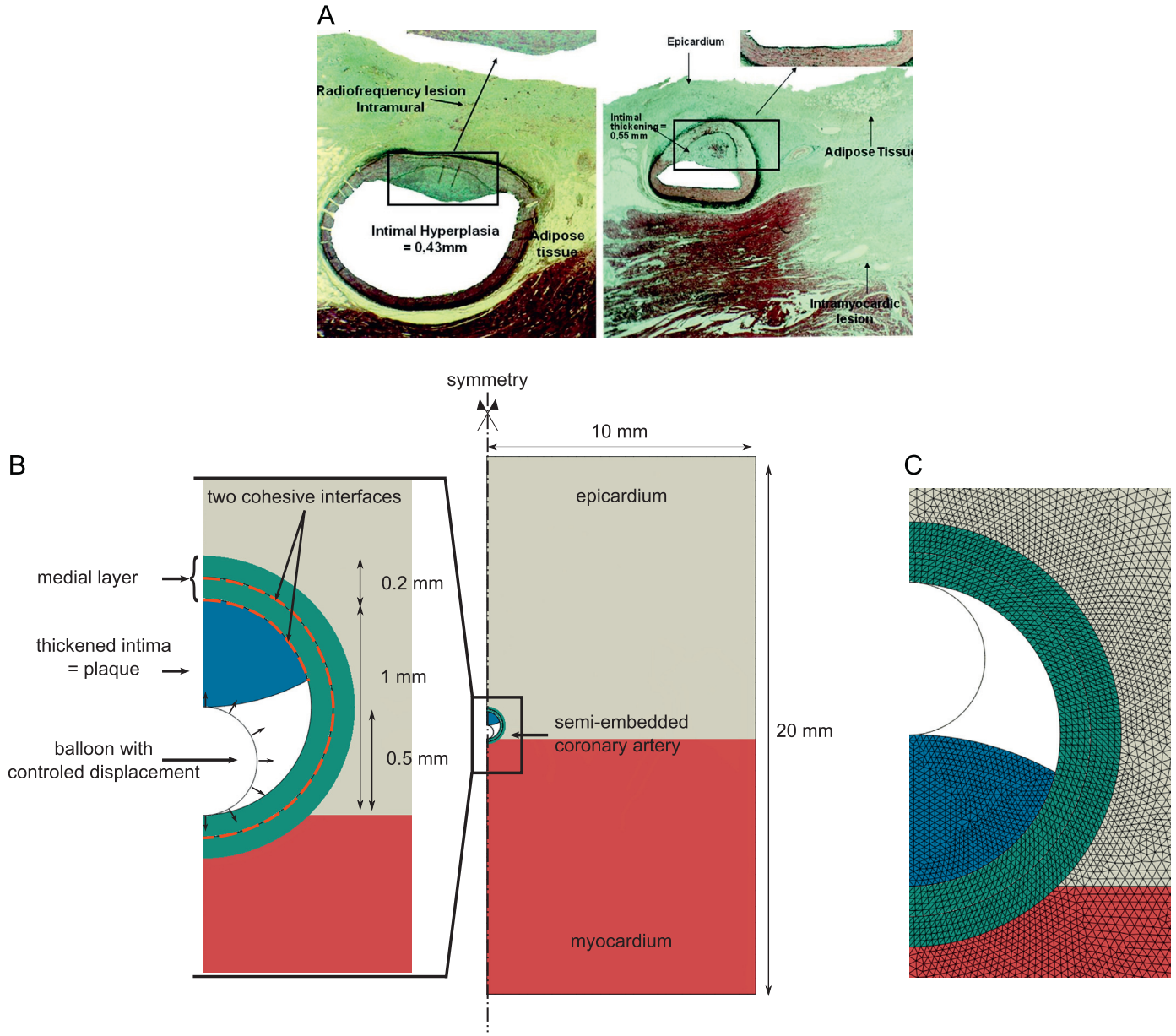

Fig. 1. (A) Histological photograph illustrating the choice of the reference geometry (reprinted with permission from (Viles-Gonzalez et al., 2011)); (B) reference model: geometry, dimensions and boundary conditions; and (C) alternative geometry and illustration of the mesh density.

more at risk of complications during balloon angioplasty than others, depending on mechanical properties (tearing strength) of the arterial media and intima. These recent experimental data indicate that critical fracture energy (Gc) is significantly greater for intima than for media in LAD coronaries from patients with non-ischemic cardiomyopathy. We have also shown in mouse models that adhesion strength of plaques depends on plaque composition (Wang et al., 2013a), so it is reasonable to suggest that tearing strength of human coronary artery layers may vary among individuals depending on specific pathology (an obvious example would be connective tissue disorders such as Marfan's syndrome (Pratt and Curci, 2010)). Less information is available regarding realistic parameters for Mode II failure, although in some cases this seems to predominate.

Our recent experimental studies have suggested that we re-visit the question of dissection mechanisms during coronary angioplasty. Depending on the respective adhesion or tearing strengths of the arterial constituents in each layer, it is hypothesized in this study that dissection or failure mechanisms may vary during balloon angioplasty. The present paper focuses on the early stages of angioplasty prior to the evolution of inelastic damage in the adjacent, lesion-free wall. The aim is to study what the dissection mechanisms are and especially how they are triggered.

To address this question, our approach entailed a parametric computational study to investigate the relative importance of intimal and medial tearing strength on tissue failure mechanisms in an epicardial coronary artery undergoing balloon angioplasty. We focused our analysis on a simplified model of the LAD coronary since we have experimental data for tearing (dissection) strength of thickened intima and underlying media in terms of critical fracture energy (G_c). A finite element model was developed including cohesive interfaces to describe possible damage within the arterial wall, both at the plaque-media interface and within the media itself. Various scenarios were tested in which the