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Three-dimensional biomechanics of coronary arteries

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ABSTRACT

The focus of this paper is to model and simulate the nonlinear dynamics of atherosclerotic coronary arteries as a tool to predict the initiation of heart attack. A dynamic three-dimensional visco/hyperelastic fluid–structure interaction model of an atherosclerotic coronary artery is developed by means of the finite element method (FEM) using ANSYS. Simulations are undertaken using the model to examine the risk of plaque rupture with the following parameters taken into account with varying levels of stenosis: physiological pulsatile blood flow; tapered shape of the artery; viscoelasticity and hyperelasticity of the artery wall; effect of the motion of the heart; active artery muscle contraction; the lipid core inside the plaque; three layers of the artery wall; non-Newtonian characteristics of the blood flow; and micro-calcification; this paper is the first to incorporate all these effects. The generated model can potentially be used as a predictive tool for plaque rupture to identify the conditions that are high risk for atherosclerosis plaque rupture.

1. Introduction

1.1. Impact of this investigation

According to the World Health Organization (WHO), coronary heart disease (CAD) and stroke are the *two leading* causes of death globally with more than 15 million deaths in 2015 (WHO, 2017). This situation has been reported to be similar in Australia with CAD and cerebrovascular diseases being ranked as the first and third most important causes of death respectively with approximately 20,000 and 10,000 deaths attributed to them in 2014 (AIHW, 2017). Atherosclerosis is the predominant cause for both coronary and cerebrovascular diseases. The acute events are usually caused by plaque rupture which releases thrombogenic material into the artery lumen leading to clot formation. Identification of the vulnerable plaque, at high risk for rupturing, remains the Holy Grail in the field.

This study aims at developing a dynamic model for coronary arteries to predict the vulnerable atherosclerotic plaque that is at high risk for rupturing. This model could result in identification of patients who are at high risk of myocardial infarction and allow interventions to stabilise the plaque and prevent myocardial infarction.

Although it is accepted that increasing low-density-lipoprotein (LDL) is responsible for shaping and growing plaque and hence atherosclerotic arteries, the primary cause for plaque formation is disruption of the endothelial layer (which is the inner layer of arteries consisting of one layer of cells). A very small crack or injury in the endothelium in addition to

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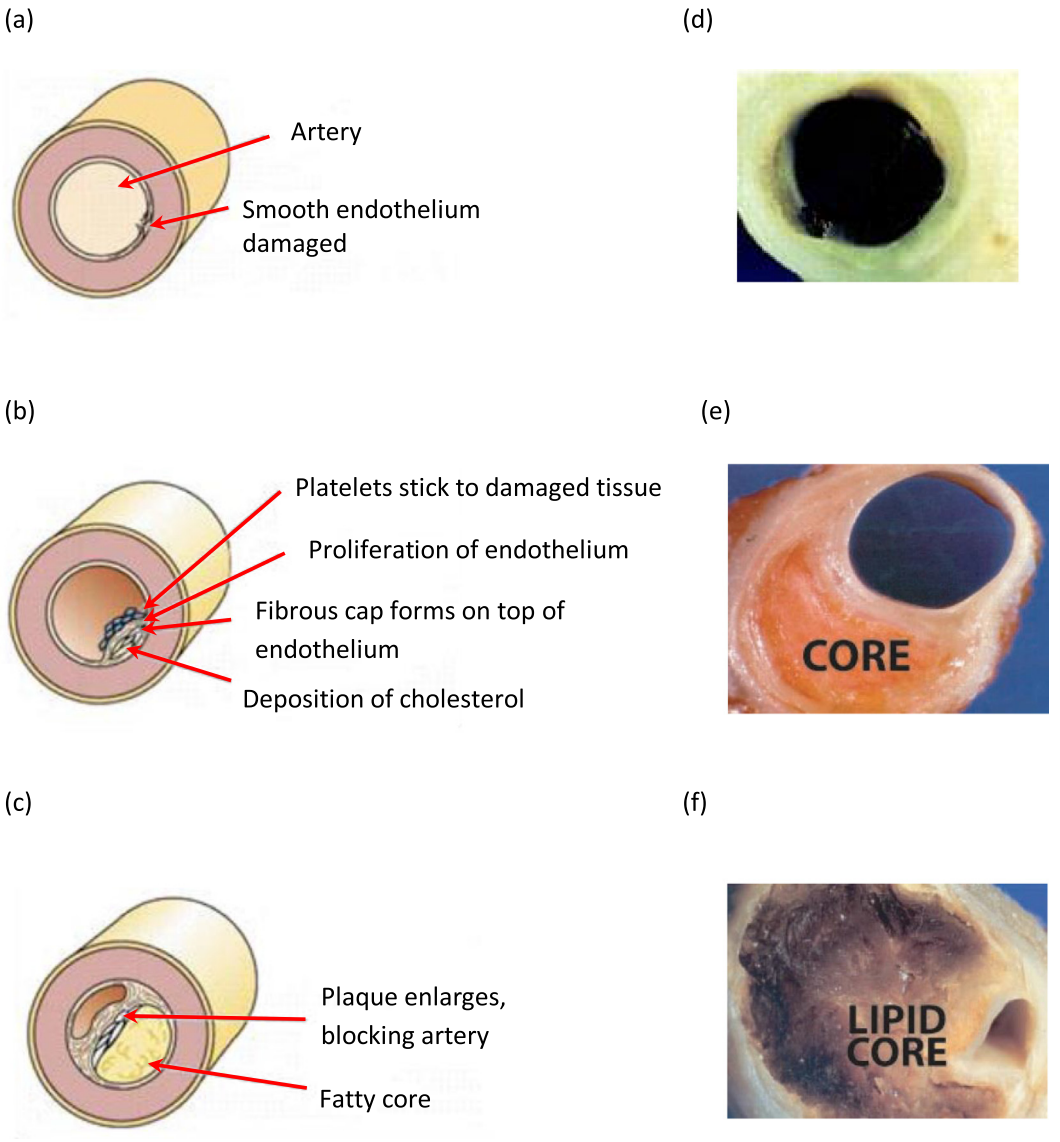

Fig. 1. Different stages of an atherosclerosis (Purves, Purves, Orians, Sadava, & Heller, 2003); (a) damaged endothelium; (b) cholesterol deposition; (c) enlarged plaque; (d–f) are the experimental (Doherty et al., 2004) counterparts of (a–c), respectively.

circulating LDL lipoprotein is thought to initiate the process of atherosclerosis. As a result, fatty streaks are seen as early as 6 months of age (Ross, 1999). These fatty streaks accumulate lipids and grow into a plaque, enlarging into the inner layer of the arteries in the course of time, and producing stenosis in the arteries. The myocardial infarction occurs due to the plaque rupture. As such, the thrombogenic materials (such as lipid and calcium) inside the plaque is released leading to thrombus formation by platelets in the blood. The thrombus obstructs the artery and the blood flow is cut from that part of the heart which causes a heart attack. Fig. 1 shows the different stages of atherosclerosis involving (a) the damaged endothelium (b) cholesterol deposition, and (c) the enlarged plaque.

1.2. Contribution of the current study to the field

Different imaging techniques are employed to investigate coronary plaques; however, it is almost impossible to predict the vulnerable plaque that is more likely to rupture. According to experiments (Falk, Shah, & Fuster, 1995), most of the ruptures occur in the stenosis range of 45–50%; this implies that the rupture is very likely to occur even when the stenosis is less than half of the cross-section of the artery. This indicates the need for the development of a computer model which simulates atherosclerosis in coronary arteries to predict the risk factors associated with the plaque rupture and resultant acute myocardial infarction. Being asymptotic is the main problem of the plaque composition which could be related to the