INFLUENCE OF INTRALUMINAL THROMBUS MATURATION ON THE ABDOMINAL AORTIC ANEURYSM ENLARGEMENT

Lana Virag[1], John S. Wilson[2], Igor Karšaj[1], Jay D. Humphrey[2]

[1] *Faculty of Mechanical Engineering and Naval Architecture, University of Zagreb, Croatia;-*

[2] *Department of Biomedical Engineering, Yale University, USA*

Introduction

Approximately 80% of abdominal aortic aneurysms (AAA) have included an intraluminal thrombus (ILT). These thrombi have complex natural histories and structures, and their biological and mechanical properties influence growth and remodeling of AAAs. The idea that ILT can change stress distribution in aortic wall is not new, but there is increasing recent evidence that ILT is also biologically active and should not be treated as homogenous inert material. We present you the first attempt to comprehensively model G&R of the thrombus-laden AAAs.

Methods

The model has to be able to describe behavior of aortic wall, formation, development and deformation of ILT, and interaction between the two, i.e. diffusion of molecules, primarily MMPs and plasmin, through ILT and wall. G&R of aortic wall is based on [Karšaj, 2012]: all three layers (intima, media, and adventitia) are modeled and continuum mixture theory is used, since each layer consists of different structurally significant constituents (elastin, SMC, collagen). The mass of each constituent changes in time, depending on quantity of their production and removal, [Humphrey, 2002]. In contrast to previous study we also model degradation of elastin and inflammation, as they are important part of aneurysmal growth. Model of formation of ILT and evolution of its mechanical properties is based on experimental results describing ILT structure, mechanical and histological characteristics (e.g., [Tong, 2011]). The basic assumption is that inner diameter of AAA under constant outer load does not change. That means that in every time step in which AAA grows, a new part of ILT is formed. Thrombus consists of fibrin mesh, and entrapped cells from blood in luminal layer. Intermediate layer is devoid of cells, while fibrin mesh in abluminal layer is mostly disorganized and degraded. Fibrin is produced by cleaving fibrinogen into fibrin by thrombin, while it is degraded by plasmin. It is important to note that inflammation, and therefore plasmin and proteases have two sources: luminal layer of the ILT and increased *vasa vasorum* in the media and adventitia in the wall. The importance of certain source changes with time: in small AAAs where luminal layer is adjacent to the wall, proteases come mostly from ILT, whereas in big aneurysms with ILT several centimeters thick proteolytically active luminal layer almost has no influence. Current model is limited to axisymmetric, cylindrical geometry.

Results and discussion

Figure 1 shows simulated growth and deformation of AAA with ILT after initial insult, degradation of 5% of mass of elastin. After luminal layer was fully formed, AAA temporarily stopped growing, a phenomenon known as step-wise growth that occurs in 65% of patients, [Kurvers, 2004]. Average rates of growth of AAA and changes in mass of wall constituents (not shown) met the expectations from experimental results in literature. It is known that AAAs are characterized by appreciable thinning of media, which is noticeable in Figure 1 from comparison of initial thickness and one during the stall.

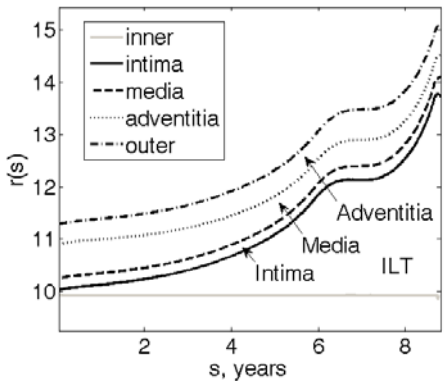

Figure 1: Growth of AAA during first 8.5 years after initial insult

References

Humphrey *et al.*, Math Models Meth Appl Sci, 12(3):407–430, 2002.

Karšaj *et al.*, Mech mater, 44:110-119, 2012

Kurvers *et al.*, J Am Coll Sur, 199(5): 709-15

Tong *et al.*, Eur J Vasc Endovasc Surg, 42(2): 207-219, 2011