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Impact of Plaque Erosion on Stress/Strain and Flow Shear Stress Calculation: An OCT-Based FSI Modeling Study

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Abstract: Plaque erosion, one of the primary causes for coronary thrombosis, is responsible for about one third of the patients with acute coronary syndrome (ACS) [1]. Histological studies characterized the eroded plaque as a plaque with following morphological features: 1) plaque intima having direct contact with intraluminal thrombus due to the absence of endothelium or endothelial injury; 2) without rupture in the fibrous cap; 3) abundance of proteoglycans and smooth muscle cells on the luminal surface under the thrombus [2]. These characteristics has been applied in *in vivo* diagnosis of plaque erosion using optical coherence tomography (OCT) imaging technology and specific treatment strategy has also been developed for plaque erosion related to ACS [3]. However, the pathogenesis mechanisms that cause plaque erosion are not fully understood [2]. It is postulated that plaque erosion has close association with biomechanical conditions in the coronary vessel [2]. In this work, a patient-specific OCT-based modeling approach was developing 3D fluid-structure interaction (FSI) models to investigate the biomechanical conditions in eroded plaque and investigate the impact of erosion-induced inflammation on plaque stress/strain conditions.

Patient-specific OCT data of eroded coronary plaque were acquired from one male patient (age: 64) with frequency-domain OCT C7XR system and the Dragon Fly catheter (Lightlab Imaging/St. Jude Medical). Erosion sites were identified as the luminal surface having direct contact with intraluminal thrombus on the OCT images. Biplane X-ray angiographic data (Allura Xper FD10 System, Philips, Bothel, WA) were also acquired with the information of the location of the coronary artery stenosis and vessel curvature. OCT images were segmented manually with external elastic membrane (EEM) contour assumed to have positive remodeling ratio 1.1 for this plaque. The trailing edge of the lipid-rich necrotic core (lipid) was also assumed since it cannot be seen on the OCT images. Co-registration of OCT and angiographic data were performed to reconstruct the 3D coronary geometry. Our FSI modeling procedures with pre-shrink–stretch process and anisotropic material properties were previously developed. Blood flow was assumed to be laminar, Newtonian and incompressible, with arbitrary Lagrangian–Eulerian formulation of the Navier–Stokes equations used as the governing equations. The governing equations of the structure models included equations of motion, the nonlinear Cauchy–Green strain–displacement relation and Mooney–Rivlin material properties. To reflect tissue weakening caused by erosion-induced inflammation, the material stiffness of plaque intima at the erosion site was adjust to one tenth of un-eroded fibrous plaque tissue [2]. Three FSI models were constructed to investigate the impacts of erosion-induced inflammation and lipid component on plaque stress/strain conditions: M1, without erosion (this means plaque intima at the erosion sites were not softened) and without inclusion of lipid component; M2, with erosion but no lipid component; M3, with erosion and inclusion of lipid component. Pulsating pressure conditions were prescribed at the inlet and outlet of the vessel segment. Fluid–structure interaction boundary conditions including force balance and no-slip conditions were imposed to the fluid–structure interface between blood flow and vessel lumen surface. Results for plaque wall stress/strain (PWS/PWSn) and flow wall shear stress (WSS) from the FSI models corresponding to peak blood pressure were extracted for analysis.

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Tab. 1 summarizes the average values of the biomechanical factors both at erosion sites and fibrous cap overlaying lipid component for 3 models. By comparing M1 and M2 at the erosion sites, Mean PWS value decreases from 49.98 kPa to 18.83 kPa (62.32% decrease) while Mean PWSn value increases from 0.1231 to 0.1384 (12% increase) as the material stiffness becomes 10 times soft. At the fibrous cap, M3 (with inclusion of lipid) will elevates mean PWS and PWSn values by 48.59% and 16.09%, respectively. The impacts of erosion and lipid on flow shear stress were limited (<2%).

Table 1: Average values of three biomechanical factors over erosion sites and fibrous cap overlaying lipid core.

Average values of biomechanical factors over Erosion sites for 3 Models			
	M1	M2	M3
Mean PWS (kPa)	49.9759	18.8318	18.8298
Mean PWSn	0.1231	0.1384	0.1384
Mean WSS (dyn/cm^2)	101.0637	99.9383	99.4303
Average values of biomechanical factors over fibrous cap overlaying lipid for 3 Models			
	M1	M2	M3
Mean PWS (kPa)	68.3209	68.3199	101.5173
Mean PWSn	0.0870	0.0870	0.1010
Mean WSS (dyn/cm^2)	18.5260	18.4634	18.1910

To conclude, erosion-induced inflammation on blood vessel would lead to lower stress distribution but larger strain distribution, while lipid component would elevate both stress and strain conditions. This shows the influence of erosion and lipid component has impacts on stress/strain calculations which are closely related to plaque assessment.

Keywords: Plaque erosion; FSI modeling; OCT; plaque biomechanics; coronary

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References

1. Kanwar SS, Stone GW, Singh M, Virmani R, Olin J et al. Acute coronary syndromes without coronary plaque rupture. *Nature Review Cardiology* **2016**, 13(5): 257-265.
2. van der Wal AC, Becker AE, van der Loos CM, Das PK. Site of intimal rupture or erosion of thrombosed coronary atherosclerotic plaques is characterized by an inflammatory process irrespective of the dominant plaque morphology. *Circulation* **1994**, 89(1): 36-44.
3. Jia H, Dai J, Hou J, Xing L, Ma L, Liu H et al. Effective anti-thrombotic therapy without stenting: intravascular optical coherence tomography-based management in plaque erosion (the EROSION study). *European Heart Journal* **2017**, 38: 792–800.