SHEAR STRESS AND WALL STRAIN DISTRIBUTION IN HUMAN CORONARY ARTERIES IN-VIVO

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INTRODUCTION

Blood flow induced wall shear stress (SS) plays an important role in the (patho)physiology of the vascular wall [1]. Recently tools became available to study the 3-dimensional SS distribution in the coronary arteries relative to local wall morphology in-vivo [2]. In addition, local strain distribution in the wall can be measured with an ultrasound-based technique called elastography. This strain distribution can be applied to determine local properties of the vascular wall [3]. We combined SS and strain measurements in-vivo to test feasibility of determining vulnerable plaque parameters relative to SS distribution.

METHODS

In a patient enrolled in a stent study, biplane angiography and an ECG gated stepped pull back with intravascular ultrasound catheter were performed to obtain the 3D geometry of a coronary segment. IVUS images were first recorded with a sheathed catheter (CVIS), which is essential for an accurate 3D reconstruction using the ANGUS method [2]. Subsequently, another IVUS catheter (JOMED) was introduced. IVUS images and elastographic data were acquired during a continuous pullback in the same arterial segment (figure 1).

SS was determined by applying computational fluid dynamics, using the 3D reconstructed arterial segment and patient-specific





figure1: Intravascular ultrasound image from the CVIS catheter of a coronary artery (left) and the resulting wall strain measurement (right).

data [4]. The flow was measured with the JOMED catheter and blood viscosity at high shear rates was determined with a capillary viscometer. The elastographic data can be used to determine radial wall strain (WS) induced by natural pressure variations. The wall strain is estimated by performing cross-correlation analysis on radiofrequency ultrasound data obtained at two different transmural pressure levels

In both IVUS recordings corresponding landmarks like side branches, stent edge and calcified areas were used to shift and rotate the JOMED IVUS data to obtain axial and circumferential matching with the CVIS data.

RESULTS

In the first patient, stent-edge and a calcified area served as landmarks for the mapping of the two IVUS data sets in axial direction. The length of a calcified area derived from the stepped pull-back was 11.0 mm (ANGUS) versus 11.3 mm from the continuous pull-back (elastography). The center of the calcified area was used for the circumferential matching. After rotation of the JOMED data by 89°, the mean variation of the location of center of the calcified area was 22°.

The viscosity was 2.8 mPas, average peak velocity was 0.13 m/s, and the resulting mean SS in the segment was 0.47 ± 0.05 Pa. The mean WS was 0.22 ± 0.12 %. We found a significant inverse relation between SS and WS after averaging the data for each axial location: WS = -0.39*SS + 0.38 (n = 23, R = 0.55, p < 0.05). In the images an isolated plaque circumferentially surrounded by high strain shoulders was located near a high SS gradient.

CONCLUSION

The combination of ANGUS and elastography is feasible and enables to map the wall strain distribution on the 3D reconstructed artery. This enables us to. investigate relationships between shear stress and wall strain related parameters of plaque vulnerability invivo. Currently, the enrollment of patients to complete this pilot study, is in progress.

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The 3D finite element mesh of the reconstructed coronary artery with the 3D shear stress distribution in a segment (left) and matching strain distribution (right).