Wall Morphology, Hemodynamics and Wall Shear Stress in Peripheral Arterial Disease

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Introduction: Lower extremity peripheral arterial disease (PAD) is a widely prevalent syndrome whose major cause is atherosclerosis.[1] It has been demonstrated that atherosclerosis in adults develops preferentially in arterial regions where wall shear stress (WSS) is low.[2] Wall shear forces, however, are complex and three-dimensional information on 3-directional blood flow velocities is thus needed to fully characterize WSS. The aim of this study was to combine multi-contrast morphological imaging of the peripheral arterial wall with the analysis of femoral hemodynamics (flow and WSS parameters) based on 2D phase contrast MRI with 3-directional velocity encoding.

Methods: All measurements were performed on a 1.5T MR system (Espree, Siemens Healthcare, Erlangen, Germany). Twenty-three patients with lower extremity PAD (age = 67.4 ± 13.6 years, range 23.4 to 87.3, 7 female) were included in the study. Multi-contrast plaque imaging was performed at the level of the proximal thighs by different MR sequences that generated four types of 2D images at the level of the proximal thighs: time-of-flight (TOF) bright-blood images; and proton density (PD), T1- and T2-weighted images with a black-blood technique. Turbo spin-echo sequences were used with a fat saturation preparation pulse (slice thickness = 3 mm, spatial resolution = 0.625 x 0.625 mm^2).

ECG-gated 2D phase-contrast MRI with 3-directional velocity encoding was acquired in a 2D slice orthogonal to the femoral artery (venc = 120cm/s along all direction, temporal resolution = 22.8ms, slice thickness = 6mm, spatial resolution = 1.1 x 1.5mm^2). The location of multi-contrast vessel wall images in the femoral artery was copied for data acquisition with 2D CINE PC to co-register information on the vessel wall and femoral hemodynamics

Multi-contrast plaque images were visually analyzed by a radiologist who classified the patients' femoral arteries as being normal or abnormal, according to the following categories: presence of wall thickening, presence of a lipid-rich plaque, presence of a loose-matrix plaque, and presence of a calcified plaque. Phase contrast data were analyzed using a home built software tool (Matlab, the Mathworks, Natick, MA, USA). After correction for Maxwell terms and eddy currents, the vessel lumen of the femoral artery was manually segmented for all cardiac time-frames. Based on the vessel lumen contours the total flow, pulsatility index (PI), and peak velocity could automatically be quantified. In addition, a cubic b-spline interpolation of the 3-directional velocity data was used to derive the local velocity gradient as a measure of the segmental distribution of the WSS vector along the vessel wall. Time-averaged absolute WSS, peak systolic WSS, and oscillatory shear index (OSI) were also calculated in two ways: (1) averaged over the entire vessel lumen circumference; and (2) in eight angular segments along the vessel wall.

Results: Multi-contrast plaque imaging in 23 PAD patients revealed n=10 normal femoral arteries with neither wall thickening nor signs of plaque. In the remaining 13 patients, analysis revealed abnormal femoral wall as characterized by wall thickening (n=3), loose matrix plaque (n==2), lipid-rich plaque (n=3), and calcified plaque (n=5).

For the quantitative comparison of flow and wall parameters (table 1 and figure 2, right) patients with normal femoral artery walls were considered as controls. As summarized in table 1, pulsatility index, peak systolic blood flow velocities and oscillatory shear index (OSI) were similar for controls and patients with femoral wall thickening or plaque. Total flow over the cardiac cycle was significantly ($p<0.05$) reduced in plaque patients. Similar, WSS and peak WSS were lower compared to controls but did not reach significance.

A more detailed regional comparison of peak systolic WSS between controls and the different subgroups of patients with femoral wall abnormalities is shown in figure 2 (right). Except for patients with loose matrix plaques, patients with atherosclerotic lesions generally demonstrated lower peak systolic WSS. In addition, ore heterogeneous segmental WSS distribution was observed in patients versus controls.

	PI	peak systolic velocity [m/s]	flow [ml/s]	WSS [N/m^2]	peak WSS [N/m^2]	OSI [%]
controls (n=10)	4.2 ± 2.5	0.73 ± 0.26	4.1 ± 1.2	0.79 ± 0.51	1.13 ± 0.66	29 ± 9
plaque (n=13)	4.3 ± 1.7	0.72 ± 0.28	2.9 ± 1.4	0.67 ± 0.31	0.98 ± 0.55	29 ± 4

Table 1: Descriptive statistics for of flow and wall parameters for patients with normal femoral arteries (controls) and patients with wall thickening or femoral artery plaque at the location of 2D CINE PC imaging.

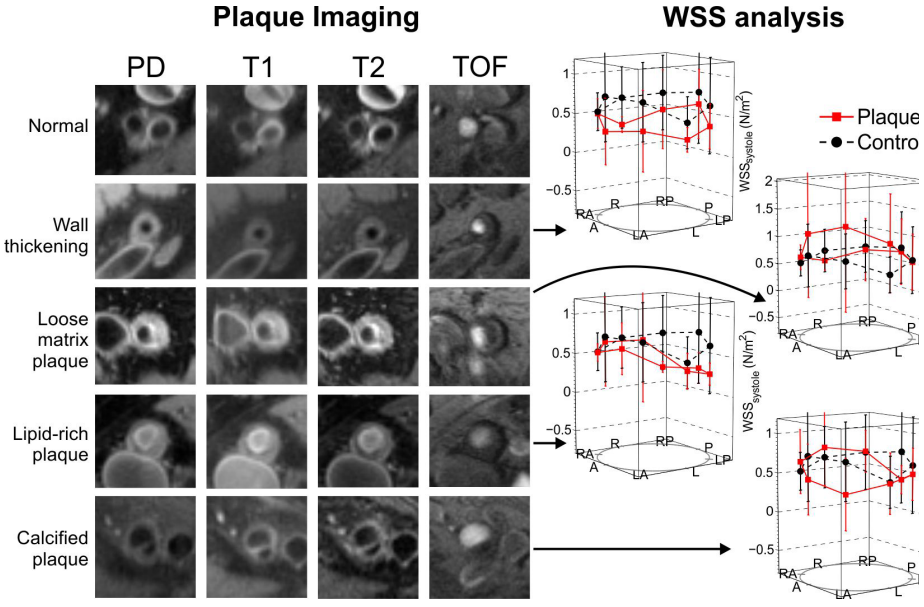

Figure 1: Example images for multi-contrast plaque characterization for a normal femoral artery (top row)

Discussion: This is the first study where both wall morphology and femoral hemodynamics are investigated in a small cohort of patients. Previous studies have shown that reduced WSS is linked to altered endothelial function and the development of atherosclerosis. A proposed mechanism is an enhanced mass transfer through the arterial endothelium, leading to a gradual accumulation of material in the arterial wall.[2] Our results demonstrate decreased peak systolic WSS in the patients with plaques, thus being consistent with the actual understanding of the causes of atherosclerosis. This technique has the potential to being able to investigate a direct connection between the presence and type of atherosclerotic plaques and changes in wall shear stress. Further analysis is warranted to co-locate segmental changes in WSS with the position and composition of aortic plaques. Limitations of this study include the small patient cohort with heterogeneous wall abnormalities, the absence of normal healthy controls, and the limited spatial resolution of 2D phase contrast MRI.

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