Wall Shear Stress of Swine Model with Induced Carotid Artery Stenosis

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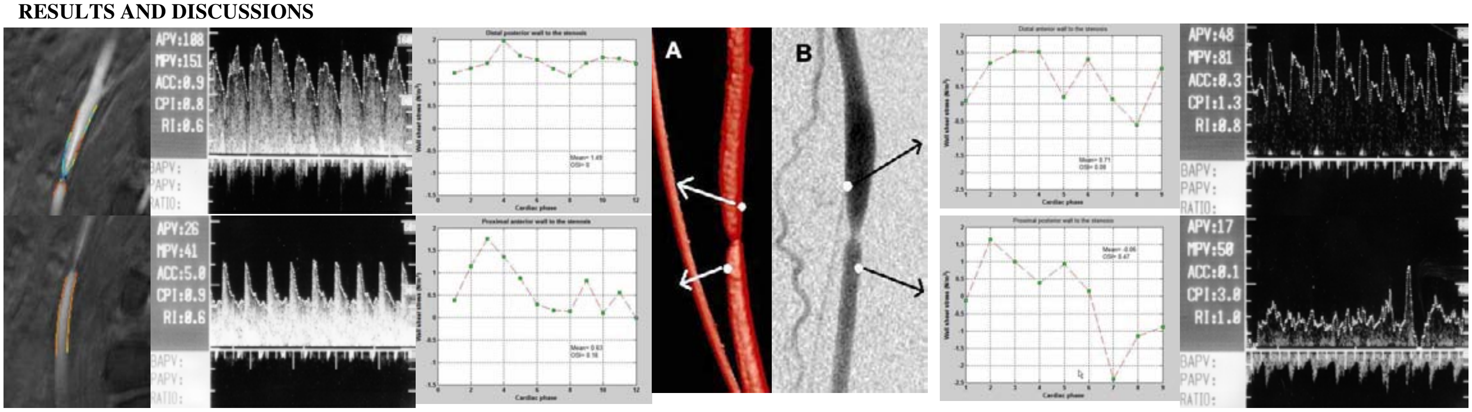
OBJECTIVES

Low and/or oscillatory shearing stress at a vessel wall is regarded as a risk factor of the evolution of atherosclerotic plaques [1]. *In vivo* hemodynamic study concurrent with temporally-resolved histo-pathological investigation has inherent difficulties especially, at the stage of formation and development of atherosclerotic lesions. In this study to estimate wall shear stress for comparison with histological results, stenosis at swine carotid artery was artificially induced by surgical partial ligation. Advanced atherosclerotic plaques were developed in a proximal portion to the stenosis. Concurrently, high-resolution MR phase contrast (PC) was performed to estimate the temporal and spatial distribution of wall shear stress within small and deeply positioned swine carotid vessel. Doppler velocimetry was used to measure and compare blood flow with MR measurement.

METHODS AND MATERIALS

Common carotid arteries were surgically ligated to cause 80% stenosis in 13 Yucatan swine. All animals were then fed with a high-fat and high cholesterol diet till euthanization. High-resolution MR-PC imaging was performed for flow velocity quantification at 1.5 T and 3 T Siemens scanners. Standard 2D RF-spoiled gradient echo sequence was used using head and neck coils with 2.2 mm slice thickness, TR/TE= 10.7/5.6 msec, 140 mm FOV, 320x320 imaging matrix, 20^o FA, 2 averages, retrospectively gated into 9~12 cardiac phases depending upon the cardiac cycle, 83.2 kHz receiver bandwidth, 64 msec temporal resolution, VENC= 120 and 55 cm/sec for SI and AP direction, respectively. Shear stress at a vessel wall was estimated by measuring the rate of shearing strain [2] at the wall using the following image post-processing steps: 1) Creation of a binary image using different contrasts of the vessel and surrounding tissues, 2) Edge detection, 3) Calculation of a tangential vector at each wall point using non-linear polynomial fitting, 4) Calculation of a normal vector as perpendicular to the tangential vector, 5) Resolved projection of 2-directional velocity components onto the tangential vector along the normal direction. Images were low-pass filtered to reduce noise and cubic-interpolated to produce continuous velocity maps, and 6) Calculation of the rate of shearing strain. Non-Newtonian fluid property of blood was neglected under the assumption that viscosity decrease by increasing the shearing rate is very small and negligible therefore, constant viscosity of 3.8 Pascal*sec was used. Several parameters were calculated to characterize wall shear stress : average, maximum, minimum, and oscillatory shear index (no oscillation, $0 \le OSI \le 0.5$, high oscillation) defined [3] as $OSI = \frac{1}{2}(1 - \frac{\tau_{mean}}{\tau_{mag}})$ where, $\tau_{mean} = \left| \frac{1}{N} \sum_{n=1}^{N} \tau_n \right|$, $\tau_{mag} = \frac{1}{N} \sum_{n=1}^{N} \left| \tau_n \right|$, and N is the number of cardiac phases. Four wall segments (proximal posterior, anterior, and distal posterior, anterior) of 6 mm each were selected to calculate the average of wall shear stress of that region and to probe into regional variations. Slice position for the distal region was differently prescribed from that for the proximal region due to a vessel curvature. Endovascular Doppler velocimetry was performed to measure blood flow and compare with MR-PC measurement. Intravascular Doppler guide wire (FloWire, VOLCANO) with 12 MHz piezoelectric ultrasonic transducer and a real-time spectrum analyzer (Flomap, Cardiometrics) were used for the measurement.

RESULTS AND DISCUSSIONS



Though surgical ligation was performed to create a concentric stenosis, vessels near the stenosis developed, varying in shape which in turn, affected blood flow. In the figure above, results of wall shear stress color-maps (± 3.1 Pascal in the model A) at the systole, superimposed onto the magnitude image, are shown. Doppler traces and temporal distributions at two regions, distal and proximal, are shown for swine model A and B. The distal region to the stenosis showed highest instantaneous wall shear stress (3 ~ 8 Pascal) which demonstrates increased blood flow passing after the stenosis. It also showed high average wall shear stress and little oscillation (mean (Pascal)/OSI: 1.49/0 in model A and 0.71/0.08 in model B). On the other hand, the proximal region to the stenosis showed low and/or oscillating shear stress (mean (Pascal)/OSI: 0.63/0.18 in model A and -0.06/0.47 in model B) where advanced atherosclerotic plaques were developed. MR-PC measurement of the flow in the swine model was validated by Doppler velocimetry which demonstrated good agreement between two methods. Estimation of wall shear stress at the stenosis was not feasible due to a small vessel dimension at the given imaging resolution.

CONCLUSION

Low and/or oscillating wall shear stress was observed at the proximal region to the stenosis induced by surgical ligation during atherosclerotic plaque formation in the swine model. The distal region to the stenosis showed relatively high wall shear stress with little oscillation, depending upon the vessel geometry. Calculated wall shear stress based on PC-MRI datasets was consistent with Doppler velocimetry measurement. High resolution MR-PC imaging in conjunction with the swine model may be beneficial to providing an in-depth understanding of the hemodynamic effects on the evolution of atherosclerotic lesions.

REFERENCES

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