P83 A pilot study to assess peak systolic velocity as a possible marker of atherosclerotic burden using ultrasound

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P83: A PILOT STUDY TO ASSESS PEAK SYSTOLIC VELOCITY AS A POSSIBLE MARKER OF ATHEROSCLEROTIC BURDEN USING ULTRASOUND

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Results: 5 female SCAD patients and 9 healthy controls (C) were enrolled (age 45 ± 9 vs 45 ± 13years, p = 0.95; BMI 21 ± 3 vs 23 ± kg/mq, p = 0.22; mean BP 77/C6 vs 85/C6 mmHg, p = 0.053). 2nd interface peak was reduced in the SCAD group (97 ± 29 130 ± 19, p = 0.04), whereas RMSE/mean was increased (1.89 ± 0.68 vs 0.97 ± 0.30, p = 0.02). Similar values were found for the 1st interface. IMT (0.165 ± 0.031 vs 0.125 ± 0.022mm, p = 0.03), but not AT (0.095 ± 0.020 vs 0.081 ± 0.020mm, p = 0.20) and IMAT (0.260 ± 0.049 vs 0.206 ± 0.030mm, p = 0.053), was significantly higher in SCAD.

Radial internal diameter and wall/lumen ratio were similar: conversely WCIA was increased in SCAD (1.69 ± 0.48 vs 1.07 ± 0.37mm², p = 0.02).

Conclusions: Radial arteries of SCAD patients were characterized by increased wall thickness. Furthermore, the 2nd echogenic layer exhibited loss of echogenicity and inhomogeneity, features similar to FMD patients.

Conclusions: A method for asymptomatic carotid plaque characterization using CEUS is presented. We focused on plaque lipid variations and the possible influence of statin therapy. We showed that carotid plaques are rarely stable, but rather continuously change composition over time and how statins could play an important role in this process.

References

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IMAGE-BASED CHARACTERIZATION OF PLAQUE LIPID CONCENTRATION CHANGES IN TIME AND THE ROLE OF STATIN THERAPY
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Introduction: Carotid artery atherosclerosis is an established risk factor for cerebrovascular events. Core lipid-rich plaques are considered at a higher risk of embolization compared to fibrous or calcified lesions. Contrast enhanced ultrasound (CEUS) is effective for studying carotid plaques, providing a virtual histology [1]. Here we assess the behavior of non-surgical carotid plaques in terms of lipid variation over time.

Methods: Eleven patients were enrolled (University of Turin) with a 50–69% (ECST) carotid asymptomatic stenosis. Seven patients were on statin therapy. All patients signed an informed consent and underwent standard carotid ultrasound (MyLab25 Gold, Esaote). A 1.5ml bolus of SonoVue (Bracco) was injected; then a 5ml saline flush. Post-contrast Bmode images (180s after injection) were saved and analyzed offline. All patients repeated this protocol after 6 months.

The plaques were segmented, intensity normalized [2], and characterized according to a previous method [3].

Results: We evaluated small cohorts according to lipid concentration changes, identifying four categories. One patient had a plaque showing no lipid variation; four patients showed a slight decrease; four patients a remarkable decrease; two patients an increase.

Seven patients with a decrease in lipid concentration were on statin therapy, while both patients with an increase in lipids were not.

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A PILOT STUDY TO ASSESS PEAK SYSTOLIC VELOCITY AS A POSSIBLE MARKER OF ATEROSCLEROTIC BURDEN USING ULTRASOUND
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Introduction: Ischemic heart disease (IHD) has been associated with lower peak systolic velocity (PSV) on penile Doppler measurements [1]. This study establishes whether carotid ultrasound (US) PSV was associated with computational fluid dynamics (CFD) outputs, which in turn may contribute to IHD pathogenesis.

Methods: A sample of 57 subjects (with IHD: 27, without IHD: 30) had US velocity profiles (left- common carotid artery) determined between 10–12 equi-spaced points. Bezier curve fitting was used to fit the profile through the measured velocity points for a normalised diameter. PSV was correlated against CFD results such as wall shear stress (WSS) [2]. Difference in PSV between individuals with/without IHD was studied via t-test. Linear regression was carried out to see if peak systolic velocity was associated with CFD outputs. Any significant associations were analysed within stratified groups (with/without IHD).

Results: PSV was significantly lower (p = 0.042) in subjects with IHD (with IHD: 53.6 ± 17.3 cm/s, without IHD: 62.8 ± 16.1 cm/s). PSV was associated with carotid bulb average pressure drop (p < 0.001), area of average bulb WSS (<1 Pa: p = 0.016, <2 Pa: p = 0.006, <3 Pa: p = 0.001). All the above associations remained significant in individuals with IHD (average bulb pressure drop: p = 0.001, average bulb WSS (<1 Pa: p = 0.013, <2 Pa: p = 0.008, <3 Pa: p = 0.003). In subjects without IHD, PSV was associated with only average bulb pressure drop (p = 0.016).

Conclusions: This study suggests that further work on PSV and its associations with CFD outputs is required in individuals with and without IHD in various vascular beds.

Reference

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HIGH-FRAME RATE VECTOR FLOW IMAGING: RELATIONSHIP BETWEEN CAROTID BIFURCATION GEOMETRY AND FLOW PATTERNS
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Background: The laminar flow movement in straight arteries is affected by anatomical factors such as bifurcation, lumen diameter changes or plaques.

Methods: The study was set up to establish an approach for a non-invasive and low-cost tool for evaluating the relationship between geometry of the carotid bifurcation and flow patterns. To this end, a mathematical model was created, taking into account the realistic geometry of the carotid bifurcation. Subsequently, the model was used to simulate the flow field and to identify the key parameters affecting flow patterns.

Results: The simulations showed that the geometry of the carotid bifurcation has a significant impact on flow patterns. In particular, changes in lumen diameter at the bifurcation point, as well as the presence of plaques, were found to significantly alter the flow field. These findings are consistent with previous studies and provide new insights into the relationship between geometry and flow patterns.

Conclusion: The developed approach offers a promising tool for evaluating the relationship between carotid bifurcation geometry and flow patterns, which can have implications for understanding the development and progression of atherosclerosis.

References