High Resolution 3D Carotid Plaque Perfusion Mapping and Its Association with T2 Hyperintensity

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Introduction
Commonly applied MRI criteria for carotid plaque inflammation are an increased T2-weighted signal intensity (SI) and/or enhancement after gadolinium injection. The observation of hyperintense regions on T2 images, matched by increased contrast enhancement may identify lesions with particularly high risk to cause thromboembolic events. The transfer constant ($K_{trans}$) of contrast material into the extracellular space is a validated marker of plaque neovascularization. The association between $K_{trans}$ and increased SI on T2 images of carotid plaque remains unknown, and was investigated in this study.

Methods
Carotid MRI was performed as part of an IRB-approved protocol on 11 patients in a 3.0T clinical scanner with a custom 8-channel carotid coil described previously in [1]. Patients were recruited based on the results of a clinically indicated carotid ultrasound exam. For MRI, T2-weighted images were acquired with a 3D SPACE technique (T2w-SPACE, a variant of 3D-TSE) [2] with high spatial resolution (0.7mm isotropic). Signal intensity in the carotid wall and in carotid plaque was categorized as normal (within 50% of the surrounding tissue intensity), medium, and high (> 200% of surrounding tissue). Dynamic contrast enhancement was measured with a 3D, inversion-recovery-prepared gradient echo sequence with 0.8 mm in-plane resolution, using a gadolinium contrast bolus of 0.1 mmol/kg injected at a rate of 0.5 ml/s. The perfusion sequence parameters were: TR/TE/flip = 4.9/2.3 ms /20º; effective TI = 50 ms; matrix size: 256 x 218; rFOV = 190 x 160 mm; section/slice thickness = 3 mm; slice resolution = 56%; parallel imaging (GRAPPA) acceleration factor of 2; acquisition time per 3D stack/temporal resolution: 8 s. The plaque locations were identified from multiple reformatted views of the 3D SPACE images. The axial location of the plaque on the SPACE images was matched with a corresponding slice of the perfusion data set. The inside and outside contours of the carotid wall, including all plaque, was manually traced on each image, followed by rigid registration to eliminate any residual motion between frames. An arterial input function was generated from a region of interest in the center of the vessel lumen. $K_{trans}$ was determined for each voxel between the two traced contours, using a Kety-Schmidt-model-based deconvolution, with an additional term for the vascular signal component within each voxel.

Results
Regions with high signal intensity on T2 SPACE consistently co-localized with regions with $K_{trans}$ above the normal range (example in Figure 1). Spearman's rank correlation $\rho$ between $K_{trans}$ and the T2w signal intensity category was 0.75 (95% CI: 0.55 – 0.87). $K_{trans}$ was significantly higher in regions with medium or high signal intensity on T2-SPACE images, compared to regions in the carotid wall with normal signal intensity, as shown in Figure 2. Furthermore within larger plaques distinct sub-regions could be identified on the $K_{trans}$ maps, suggesting that heterogeneity of $K_{trans}$ within plaques may be identifiable, using a 3D image acquisition with high spatial resolution.

Conclusions
The association between $K_{trans}$ and hyperintensity of T2-SPACE images confirms by MRI a close relationship between markers of inflammation and neovascularization in carotid plaque, respectively. Pixel-level mapping of $K_{trans}$ can identify distinct sub-regions within plaques that also show distinct features on T2-SPACE images.


Figure 1, left: Example of T2 SPACE image of plaque (red arrows) with high signal intensity in internal carotid artery. Right: A matching $K_{trans}$ map at same axial location indicates areas with highest neo-vascularization (red and light blue) co-localizing with areas of high SI on T2-SPACE images.

Figure 2: $K_{trans}$ was strongly associated with the level of signal intensity observed in the same region on T2-SPACE images, indicating agreement between markers of plaque neovascularization and inflammation, respectively.