Multiparametric T1 weighted imaging may identify carotid plaque components

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Introduction: High-resolution MR imaging of the carotid wall has been show to be capable of detecting a variety of plaque components. In particular, the use of multiparametric contrast imaging including T1W, T2W, PD, DIR and 3D TOF has been shown to detect and stage certain plaque components with high sensitivity and specificity [1,2]. However, the acquisition of these various imaging parameters is time consuming, and in their reported implementation, two dimensional. Recently, multiple T1W imaging techniques have been shown to identify various components of a vulnerable plaque: black blood T1W imaging has been shown to detect intraplaque hemorrhage [3], and contrast enhanced MR provides an indication of the extent of neovascularity [4] as well as degree of inflammation [5] within the plaque. This work investigates the potential utility of using multiple T1W images before and after the administration of a blood pool agent towards assessing plaque components, especially those associated with plaque vulnerability.

Methods: Informed consent was obtained from patients scheduled for carotid endarterectomy. Imaging was done the day previous to surgery on a 1.5T GE EXCITE (GE Medical Systems) using a dedicated phased array receive only carotid coil (ScanMed USA). A high resolution 3DT1W volume (0.5x0.5x1mm) centred at the carotid bifurcation was acquired before and after injection of 0.2ml/kg Vasovist (Bayer Schering AG) blood pool contrast agent. A low b value diffusion pre-pulse was used for flow suppression as well as a spectral inversion for fat suppression. Twenty minutes after contrast injection a delayed 3DT1W bright blood sequence was acquired over the same volume to detect any delayed enhancement from the blood pool agent. Endarterectomy specimens were collected, embedded in paraffin and stained with H&E as well as CD34 and CD68. Images of the carotid arteries were manually segmented from the volumes and three dimensionally rigidly registered using a mutual information gradient descent method. An automated k-means clustering algorithm was then run on the data with n=3-16 clusters. A trained observer identified histological areas that corresponded to intraplaque hemorrhage, fibrous cap, lipid core, neovessel rich area and macrophage rich area.

Results: Figure 1 shows a representative slice from a set of T1 weighted volumes of a patient with severe carotid disease. Figure 2 shows H&E stained slide of the common carotid artery of the same patient. Inset are areas of intense CD68 positive macrophage staining as well as regions of high vascularity derived from the CD31 stains.

Discussion: This work demonstrates an initial feasibility study using multiparametric T1W imaging to identify different components of atherosclerotic plaque and compared histological correlation to these features. These 3D volumes easily segment using a standard k-means clustering approach, delineating various components of the plaque. Surgical and histological processing of the specimens significantly distorts their architecture from their geometry in-vivo, and the histological correlation is not one to one. Most significantly, the adventitial layer is not removed during the operation and the adventitial enhancement seen in these images is not represented histologically. Good animal models of complicated plaque enhancement are required for correct histological correlation of these features. However, as shown, large regions of the histology correspond with imaging findings. The components identified in this study can be obtained using a simple black blood pre and post contrast images, along with a delayed enhancement image, significantly reducing the required exam time to accurately identify these various plaque constituents.

Figure 1: Precontrast black blood T1 slice (top left) shows a foci of intraplaque hemorrhage (green arrow), post contrast black blood T1 images (top right) delineate a ring around the vessel lumen (peach arrows), indicating possible cap enhancement as well as deep in the plaque. Additionally, there is enhancement adventitially (turquoise arrows). Bright blood delayed enhancement images (bottom left) show a region deep in the plaque that is non enhancing (blue arrows). All these arrows are overlayed on a 9 cluster kmean map (bottom right). Clusters are clearly identified and classified each of these plaque features.

Figure 2: H&E stained slide of the corresponding common carotid. Intraplaque hemorrhage is identified (green box) with the inset an enlargement of the H&E stain. Areas rich in neovessels (turquoise box) are found at the periphery of the plaque, with inset showing regions rich in endothelial lined vessels identified on CD31. Areas rich in macrophage staining on CD68 (peach boxes) including the cap, deep in the lipid core of the plaque and surrounding the neovessels. Additionally, a thick region of medial hyperplasia is seen (blue box).