Measurement of the Mean ADC Values of Lipid, Hemorrhage and overall Wall Components using in-vivo Human Carotid Artery Diffusion Weighted Imaging

S-E. Kim[1,2], S. McNally[2], L. K. Findeiss[2], J. Hulet[3], J. Roberts[1,2], E-K. Jeong[1,2], D. L. Parker[1,2], and G. S. Treiman[4,5]

[1]UCAIR, University of Utah, Salt Lake City, Utah, United States, [2]Department of Radiology, University of Utah, Salt Lake City, Utah, United States, [3]Department of Biomedical Informatics, University of Utah, [4]Department of Surgery, University of Utah, Salt Lake City, Utah, United States, [5]Veterans Affair, VASLCHCS, Salt Lake City, Utah, United States

INTRODUCTION: The development of atherosclerotic plaques is a complex process. These plaques are composed of varying degrees of lipid, necrotic tissue, loose connective tissue, hemorrhage, and calcification[1-2]. The extent of lipid accumulation and the presence of intramural hemorrhage have been found to be associated with the degree of plaque vulnerability and risk of plaque rupture. However, lipids and hemorrhage are often difficult to distinguish from other plaque components by MRI, and the reported appearance of these tissues in different MR studies is inconsistent[3-4]. Reports use a variety of different sequences and criteria for their identification and measurement. Ex-vivo studies have found that diffusion weighted imaging(DWI) may provide a tool for the characterization of these components[5-6]. . Recent ex vivo studies of carotid plaques found that DWI could detect lipids and hemorrhage with greater sensitivity than other MRI sequences[7-8]. We have reported in-vivo DWI of human carotid artery using 2D ss-IMIV DWEPI sequence[9]. This study reports the ADC values of lipid, hemorrhage and overall wall composition as measured with in-vivo DWI, with histology used as the basis for comparison.

METHODS: All studies were performed on a Siemens Trio 3T MRI scanner with our custom made four element bilateral phased-array carotid coil. 2D TOF MRA was used to locate the carotid bifurcation and determine the extent of the lesion. Carotid arteries were scanned with 2D ss-IMIV-DWEPI. The imaging parameters were described on our previous work[9]. The in-plane spatial resolution for data acquisition was 1.0x1.0mm with display resolution 0.5x0.5 mm^2, after zero-filled interpolation. The apparent diffusion coefficient (ADC) map was calculated and displayed using IDL (Interactive Data Language etc.). Sequences obtained were 2D T1w, T2w, PD, 3D MPRAGE, and DWI at the same locations. CTA was also obtained in vivo. T1w images were acquired with 2D TSE with the time efficient double inversion preparation sequence[6]. 2D T2w and PD images were acquired using the 2D TSE sequence. 3D MPRAGE images were acquired with 0.5x0.5x1.0mm^3 voxel resolution and were used to identify hemorrhage. Human carotid endarterectomy specimens were obtained and fixed in 10% buffered formalin. Following decalcification, specimens were serially cross sectioned and stained. MR images and histological slides were matched using the known location and distance between MRI and histological cross section.

RESULTS:

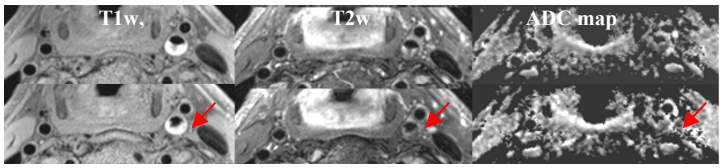

Figure 1.T1w, T2w and ADC map component

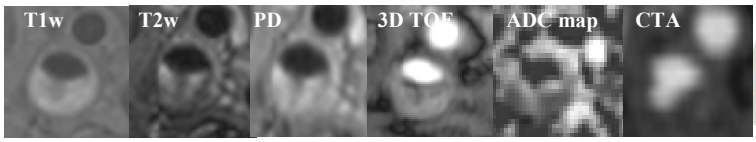

Figure 2. MRI sequences detailing carotid wall noted by the red arrows in figure 1.

Table 1 Mean value of ADC on Wall and Plaque Components

	Wall Area	Lipid Core	Hemorrhage
ADC (10^{-3} mm^2/s)	1.28±0.09	0.42±0.18	0.91±0.29

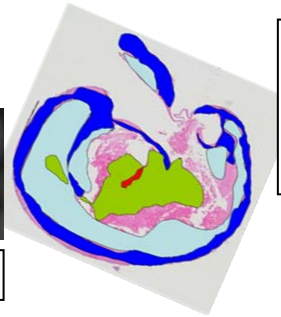

Figure 3 – Histology of the same location seen in figure 2.
Red –Hemorrhage,
Blue – Collagen Rich,
Green- Necrosis,
Light Blue – Lipid Rich Core

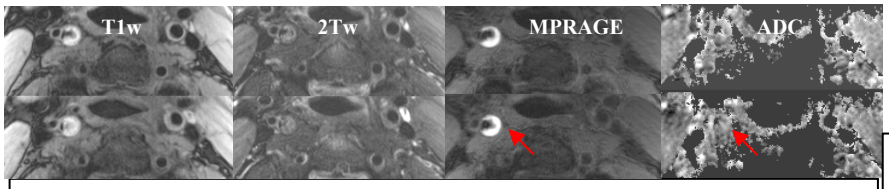

Figure 4: T1w, Tw2 images, 3D MPRAGE and ADC maps of a carotid artery with intraplaque hemorrhage.

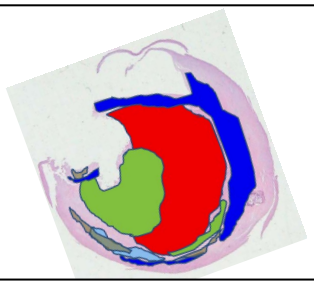

Figure 5 – Histology of the same location seen in Fig 4.
Red –Hemorrhage (1-6 weeks old), Blue – Collagen Rich,
Green- Necrosis, Light Blue – Lipid Rich Core,
Brown-Calcium

The mean ADC values of lipid, hemorrhage, and overall wall composition was calculated using ADC measurements from three different plaque locations (Table 1). Figure 1 displays the ADC map, and T1w and T2w images from this patient. Figure 2 shows T1w, T2w, PD and 3D TOF sequences at the same location indicated by the red arrow in figure 1. The ADC map in Fig 1 demonstrates clear contrast between normal and diseased arterial wall. The plaque indicated by red arrows in Fig 1 shows a bright signal on T1w, moderate signal on T2w and low ADC value (0.42x$10^{-3}$$mm^2$/s). Histology confirmed that this area represent lipid (light blue area on Fig 3). Fig 4 displays the T1w, T2w, and 3D MPRAGE images and the ADC map from a subject with intramural hemorrhage. The hemorrhage is indicated by the red arrows and shows a very bright signal on 3D MPRAGE with a moderate ADC value (0.91x$10^{-3}$$mm^2$/s). Histology confirmed that this area represent fresh hemorrhage (red area on Fig 5).

DISCUSSION: The results obtained indicate that an ADC map may be of substantial value in identifying lipid, hemorrhage, and overall wall plaque burden. Our ADC values obtained using the 2D ss IMIV DWEPI technique match previously reported ADC value in an ex-vivo study[5-8]. No in vivo studies have reported ADC values for these plaque components. This technique can potentially be applied to further investigate the ADC of other plaque components.

ACNOWLEDGEMENT: Supported by HL 48223, HL 53696, Siemens Medical Solutions, The Ben B. and Iris M. Margolis Foundation, and the Clinical Merit Review Grant from the Veterans Administration health Care System.

REFERENCES:

1. Larose E., et al. *Circulation*. 2005;**112**:2324.
2. Shinnar M, Fallon JT. et al. *Arterioscler Thromb Vasc Bioly*. 1999;**19**:2756
3. Cai J, et.al. *Circulation*. 2002; **106**:1368.
4. Toussaint JF, LaMuraglia GM, et al. *Circulation*. 1996;**94**:932
5. Clarke ES, Hammond RR, et al. *MRM*. 2003; **50**:1199.
6. Toussaint JF et al. *Arteriosclerosis, Thrombosis, and Vascular Biology*. 1997; **17**:542.
7.. Vierck J., et al. *Arterioscler Thromb VascBiol*. 2005;**25:**240.
8. Qiao, Y., et al. *Arterioscler Thromb VascBiol*. 2007;**27:**1440.
9. Kim SE, et al. *JMRM*.2009;**30**:1068
10. Kim SE, et al. *MRM*.2004;**52**:1379