## Longitudinal observation of Carotid Artery Intraplaque Hemorrhagic Volume on Magnetic Resonance Direct Thrombus Imaging

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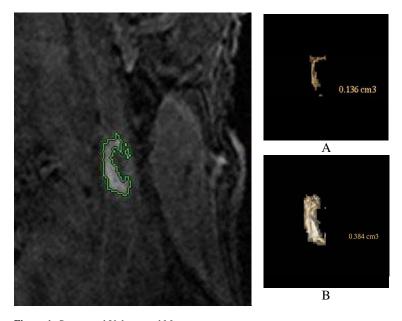
**Purpose**: Carotid artery intraplaque haemorrhage (IPH) appears to be an important marker of plaque instability leading to cerebral ischemia <sup>[1]</sup>. The change in volume of IPH may help further stratify the risk of subsequent cerebrovascular events<sup>[2,3]</sup>. Magnetic resonance imaging has the ability to detect IPH as a signal hyperintensity, however, the evolution of this signal hyperintensity is unclear. The purpose of this study was to measure IPH volume on Magnetic Resonance Direct Thrombus Imaging (MRDTI) and observe the natural evolution of IPH volume longitudinally and correlate this evolution with the presence or absence of prior ischemic events.

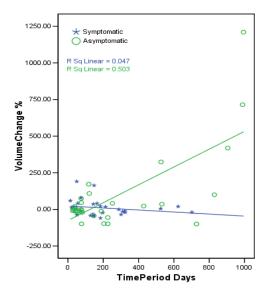
**Materials and Methods:** All patients receiving more than one clinical MRDTI scan between the periods of Jan. 2003 to Sep. 2007 were candidates for the study. Of the 140 patients receiving more than one scan during this period, 57 patients were diagnosed with complicated plaque detected by MRDTI for a total of 132 separate time points with the first scan defined as time zero. 28 patients with previous ipsilateral ischemic events were classified as symptomatic patients whereas the remaining 29 patients without prior ischemic events were classified as asymptomatic of the 3D MRDTI sequence were 6.7/1.7/15 (TR/TE/ $\theta$ ), and a reconstructed voxel size of  $0.59 \times 0.59 \times 1$  mm<sup>3</sup>. IPH volume was measured using the Advantage Workstation (GE, ADW v4.2, USA) by first cropping the image to include only the plaque and carotid arteries. Voxels from the cropped volume above 125% of the signal intensity of the spinal cord were included in the volume measurement as shown in Figure 1. Two trained observers analyzed the images to measure IPH volume. Intra- and inter-observer variability was calculated between the two data sets using Pearson's R coefficient and the coefficient of variation.

**Results** The mean time interval between scans was 181 days (Range 14~994 days). Using the methodology outlined above, intra-and inter-reader coefficient of variation for IPH volume were 6.2% and 8.2%, with correlation coefficients of 0.987 and 0.972 respectively. Relative mean change in IPH volume for asymptomatic patients was 86.31±266.44%, while for symptomatic patients, mean IPH volume change was 10.23±54.87 % (P<0.01).

## **Discussion and Conclusions:**

It appeared that symptomatic patients presenting with carotid artery IPH experienced a smaller growth in their plaque haemorrhage volume than asymptomatic patients. This indicates that asymptomatic patients may experience a significant plaque volume expansion. One possible explanation for this phenomenon is that symptomatic plaques have already reached a maximal volume and the IPH has caused plaque disruption, generating micro emboli. However, asymptomatic plaques may undergo future IPH with an increase in IPH volume. Thus, volume of IPH in association with presenting symptoms appears to be an indicator of possible future IPH growth. Further studies are required to correlate plaque IPH volume growth to patient symptoms and outcomes.





**Figure 1:** Segmented Volume and Measurement A single slice from an MRDTI volume with a signal hyperintensity at the right carotid bifurcation. The green line indicates the region selected after both a manual and a signal intensity threshold segmentation. A: 3D rendering of plaque at time =0. B: after 148 days.

## **References:**

- 1. Moody AR et al. Circulation 2003;107:3047
- 2. Yamada N. et al. AJNR 2007; 28:287
- 3. Altaf N. et al. J Vasc Surg 2007; 46:31

**Figure 2:** Plot of IPH volume change as a function of time. While over the first year no difference in IPH volume between symptomatic and asymptomatic patients is seen, beyond this, asymptomatic patients (in green circles) seem to have a dramatic increase in IPH volume.