

A CE-MRI study assessing the uptake of Gd-DTPA and the stability of atheromatous tissue within the carotid arteries

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Introduction: Inflammation is thought to be a factor associated with atheromatous plaque instability. Contrast enhanced MRI (CE-MRI) has a potential to detect inflammation within the fibrous cap and thus identify patients at high risk of thromboembolic complications due to plaque disruption. In this preliminary study, we compared the CE-MRI measurements of atheromatous plaques with histological assessment of plaque stability.

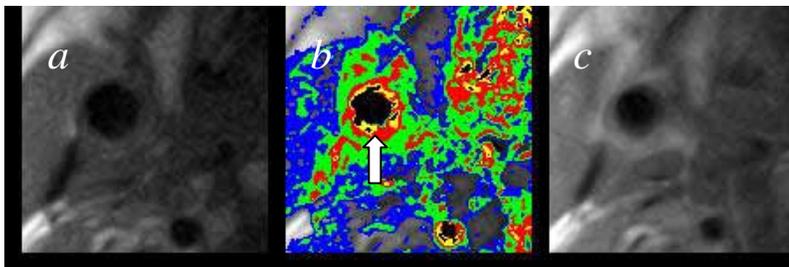
Methods: 15 consecutive patients referred for carotid endarterectomy (CEA) were recruited after formal consent had been obtained. T1-weighted images were acquired from these patients as part of an MRI examination made approximately 15 hours before the CEA. These data were acquired both before and after the i.v. administration of a weight-related standard dose (0.1 mmol/kg) of the MR contrast agent Gd-DTPA. Images were obtained with the slices centred over the carotid bifurcation using the Black-Blood technique¹ and cardiac triggering with data acquired on every heartbeat and TE/slice thickness/Field of View/Number of averages 22ms/2.7mm/120mm/5 respectively. After the CEA, the excised specimens were decalcified, sectioned and histology slides were prepared after Haematoxylin and Eosin staining. The stability of the plaque tissue was then classified either as stable or unstable by an experienced observer (CA). The CE-MRI data were then processed and assessed by an observer blinded to the results of histological assessment. The 'pre- and post-contrast' T1-weighted images were initially registered using a Normalised Mutual Information algorithm. Image processing software then delineated equivalent outlines corresponding to the vessel lumen in the pre- and post-contrast data to allow qualitative assessment of the registration efficacy. Quantitative values of enhancement (percentage SI increase over baseline) were calculated and displayed as colour overlays (Figure 1b) using software developed in-house. The signal enhancement was deemed to be 'marked' if firstly it occurred within at least half of the thickness of the atheroma and secondly, if it was found in close proximity to the lumen. The plaque tissue was only considered to be enhancing if it exceeded that measured in the surrounding healthy muscle (~40% enhancement – shown in blue on Figure 1b). A blinded comparison was made between the presence/absence of enhancement and the classification of plaque stability.

Results: Image registration was successful in 13 out of 15 studies. In two patients severe image misregistration between the pre- and post-contrast T1-weighted MR images precluded further analysis. Table 1 summarises the blinded comparison between stability and the presence/absence of enhancement in 13 CE-MRI studies and Figure 1 shows representative results obtained from a CEA patient with unstable carotid plaque tissue.

Table 1:

	Enhancement low/absent	Enhancement 'marked' – see text
Stable Plaque	4	3
Unstable Plaque	1	5

Figure 1: T1-weighted images of the common carotid artery acquired (a) before- and (c) after- the administration of the contrast agent Gd-DTPA. Calculated values of enhancement are displayed in the colour image (b) where blue represents 25-50% enhancement, green 50-100%, red 100-200% and yellow 200-400% enhancement. The arrow shows marked enhancement occurring in this unstable plaque.



Discussion: Examination of the data acquired in this study indicated that the registration technique employed was not universally successful in effectively registering the pre- and post-contrast MR images. Further, the Black-Blood imaging sequence often failed to completely 'null' the blood signal intensity, partly due to the effect of the contrast agent on the T1 of blood. This may explain the relatively high number of false positive results observed (Table 1). In spite of these problems, the techniques described in this initial study show promise for the non-invasive identification of unstable carotid plaque.

[1] Edelman R. et al. Radiology 1991;181:655-660.