

Ex vivo MRI of carotid plaque excised by endoarterectomy: correlation between T1 and histologically assessed age and degree of hemorrhage in lipid rich necrotic core

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TARGET AUDIENCE

Clinicians who are related to stroke care and diagnosis of cerebrovascular diseases
Scientists who are interested in development of atheromatous plaque and hemorrhage

PURPOSE

Lipid-rich necrotic cores (LRNC) in carotid plaque with signal hyperintensity on T1WI is associated with previous and subsequent ischemic events^{1, 2}. However, the mechanisms and duration of short T1 in LRNC are not sufficiently explained. The purpose of this study was to histologically assess the age and the amount of hemorrhage in specimen excised by endoarterectomy and to compare ex vivo MRI with histology.

METHODS

Sixteen patients who underwent carotid endarterectomy were enrolled in this study. All patients underwent preoperatively in vivo 3D inversion-recovery-based T1WI (magnetization-prepared rapid acquisition with gradient-echo: MPRAGE) with a clinically standardized parameters in which signal intensity ratio of plaque to the adjacent muscle was measured.² Sixteen specimens which removed by carotid endarterectomy underwent ex vivo MRI (T1 map) within 24 hours of removal. After MRI, specimens were fixed in formalin and sliced every 3-5mm. These sections were stained with Hematoxylin-Eosin (HE), Masson's trichrome (MT) and Antibody against Glycophorin A (protein specific to erythrocyte membrane). Sixty-four histological sections were matched with ex vivo MRI. Forty-six LRNC were included in these sections. In histological examination, the degree of intraplaque hemorrhage was graded using a scale from 0 to 3, with higher scores indicating higher Glycophorin A density; score 0 (almost none), score 1 (small amount of hemorrhage), score 2 (large amount of hemorrhage), score 3 (full of hemorrhage). Furthermore, we classified intraplaque hemorrhage using HE and MT stain into recent (many red blood cells) and chronic (no or a few red blood cell). In ex vivo MRI, all sections were imaged with 5cm surface coil on a 1.5T clinical machine (Siemens, Sonata). T1 map was obtained using simple spin-echo imaging (TR=300, 900msec, TE=13msec). T1 was measured in each region of LRNC.

RESULTS

Mean T1 in LRNC with hemorrhage (Glycophorin A score 1; 286.8msec, score 2; 226.1msec, score 3; 273.5msec) were significantly lower ($p < 0.05$, respectively) than that in LRNC without hemorrhage (score 0; 400.5msec). However, there was no significant difference in mean T1 among Glycophorin A score 1, score 2 and score 3 (**Figure 1**). Mean T1 between recent and chronic hemorrhage also revealed no significant difference (recent; mean T1 296.8msec, chronic; mean T1 247.0msec, **Figure 2**). Ex vivo T1 and signal intensity ratio on in vivo MPRAGE were well correlated ($r = -0.824$).

DISCUSSION AND CONCLUSION

The results of this study indicated that presence of intraplaque hemorrhage in LRNC was associated with high signal intensity on T1WI in ex vivo MRI. However, T1 was not changed according to the degree and age of hemorrhage. Thus, high signals of carotid plaque on T1-weighted imaging are attributed to chronic hemorrhage not only to subacute hemorrhage. Chronic accumulation of erythrocytes within an atherosclerotic plaque may represent a potent atherogenic stimulus.

REFERENCES

1. Singh N, et al. *Radiology* 2009;252:502-508
2. Yamada N, et al. *AJNR Am J Neuroradiol* 2007;28:287-292

Figure 1. Graph shows mean T1 of LRNC in each Glycophorin A score.

Figure 2. Graph shows mean T1 of LRNC with recent and chronic hemorrhage.

Figure 3. Example of LRNC without recent hemorrhage. A. in vivo MPRAGE, B. ex vivo T1 map, C. Antibody against Glycophorin A, D. Masson's trichrome. The plaque showed high signal on in vivo MPRAGE and low T1 on ex vivo MRI (T1=18msec). Histologically, intense staining for Glycophorin A in erythrocyte membrane was shown. However, the plaque comprised cholesterol clefts and necrotic debris without red blood cells in Masson's trichrome stain.

