INTRODUCTION:
Atherothrombotic intracranial disease is a major cause of cerebral ischemia. Previous studies have shown that high resolution magnetic resonance imaging (HR-MRI) can be used to depict plaques on intracranial arterial walls [1]. [2] reported that plaque with post-contrast enhancement may be related to inflammation. Studies in patients with extracranial carotid disease have shown that magnetization prepared rapid gradient echo (MPRAGE) can reliably identify intraplaque hemorrhage, and may be a better predictor of clinical events than traditional radiographic methods such as percent stenosis [3]. Yet, use of MPRAGE in imaging intracranial arteries was reported in one individual case [4]. There is no report mentioning the relationship between signal intensity changes in HR-MRI and MPRAGE in intracranial lesions. In this study, we propose to use HR-MRI combined with MPRAGE to identify intraplaque hemorrhage/thrombus and plaque enhancement in patients with stenosis in the intracranial arteries.

MATERIAL AND METHODS:
10 patients (male, mean age of 55.1 years old, range from 39-70) suspected to have intracranial artery stenosis based on previous radiographic examinations (MRA, CT or ultrasound) were recruited for the study. The study was IRB approved and informed consent was obtained from all patients. Imaging was performed on a 3T MRI scanner (Magnetom Verio, Siemens, Germany) using a 32-channel head coil for signal reception. The protocols used in the study were: (1) localizer; (2) routine clinical scans of the brain that include T1-FLASH, STIR, T2-FSE, DW-MRI, ~ 15min; (3) 3DTOF (spatial resolution 0.35 x 0.4 x 0.46mm 3), ~ 6.5min; (4) T1w-SPACE (a 3D TSE variant) pre-contrast (0.5mm isotropic, 10min) [5]; (5) MPRAGE (0.7mm isotropic, ~6min); gadolinium was then hand injected, followed by (6) T1w-SPACE post-contrast (0.5mm isotropic, 10min). The scans (3) – (6) were 3D HR-MRI protocols aimed for the intracranial arteries. Care was taken to make sure that the imaging slab covered the mid cerebral arteries (MCA), basilar artery (BA) and the petrous internal carotid arteries (ICA).

RESULTS:
In the 10 patients, there were 3 situations: (1) the lesions did not show post-contrast enhancement, nor signal increase in MPRAGE, 0 case; (2) strong signal hyperenhancement in MPRAGE but the corresponding location from T1w-SPACE post-contrast images did not show obvious enhancement, 1 case (figure 1); (3) signal enhancement was observed in T1w-SPACE post contrast, but no corresponding enhancement was observed in MPRAGE, 9 case (figure 2). In these 9 cases, not every lesion is enhanced.

DISCUSSION:
Preliminary results here showed that (1) contrast enhancement did not happened in all intracranial lesions; (2) intracranial lesions with hyperenhancement in MPRAGE may be suggestive of intraplaque hemorrhage/thrombus (based on previous studies); (3) contrast enhancement of the lesion may not have associated hyperenhancement in MPRAGE, suggesting that T1w-SPACE and MPRAGE provide different information about the plaque and may complement each other in the diagnosis of intracranial atherosclerosis. Use of T1w-SPACE pre- and post-contrast combined with MPRAGE will be a very useful tool to help identify vulnerable plaques in intracranial lesions.

REFERENCES:

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