3D high resolution MPRAGE helps identify intraplaque hemorrhage in patients with intracranial atherosclerotic diseases

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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).

Image analysis was performed on a workstation (Leonardo, Siemens, Germany). Maximum projection reconstruction (MPR) was used for visualization. 3D image sets from T1w-SPACE (pre- and post-), MPRAGE and TOF were co-registered (two at a time) to relate vessel abnormalities found from different protocols using commercial software (Syngo FUSION, Siemens, Germany). MPR images perpendicular to the long axis of the arterial wall were generated for visualization. Vessel wall images were evaluated by visual inspection of the signal intensity of the intracranial atherosclerotic plaque.

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 Tw1-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.

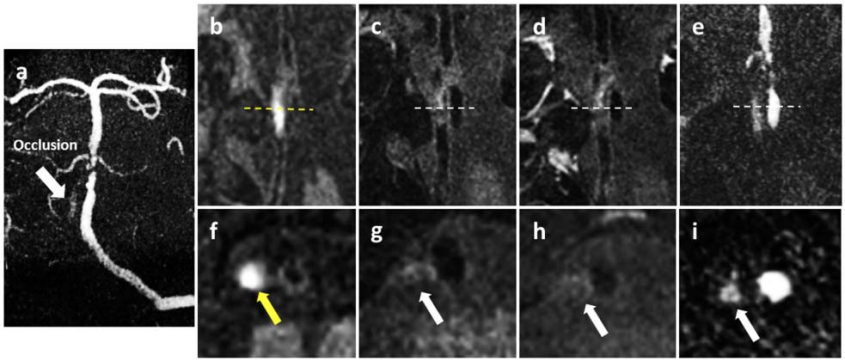

Figure 1. Images from a 55 years old patient with suspected thrombus at the right vertebral artery. (a) TOF MIP shows the complete disappearance of the right vertebral artery; the MPRAGE images (b, coronal; f, short axial) show high signal intensity (yellow arrow) at the focal area, indicating a thrombus at the stenosis; pre-Gd SPACE (c, g) and post-Gd SPACE (d, h) show the lesion at the corresponding location. And it did not enhance after Gd administration; 3DTOF (e, i) shows weak signal at that region.

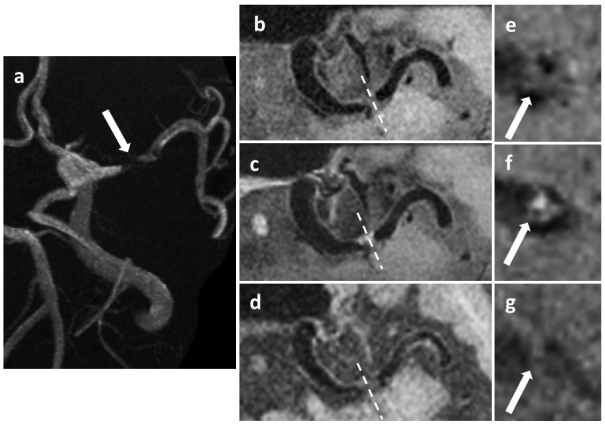

Figure 2. TOF MIP images in (a) demonstrates severe stenosis at M1 segment of the left MCA. Pre-Gd SPACE images (b, transverse; e, short axial) show an eccentric plaque at the stenosis seen in (a); the plaque is enhanced after Gd administration (c, f); however MPRAGE images (d, g) show no hyperenhancement of the lesion.

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.

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