# Comprehensive Vessel Wall Imaging for Concomitant Extracranial and Intracranial Atherosclerotic Plaques in Symptomatic Patients Using Fast 3D Multicontrast Black-Blood MR Imaging Sequences

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**Introduction:** Both intracranial and extracranial atherosclerotic vulnerable plaques are associated with cerebrovascular ischemic events. Atherosclerosis, as a systemic disease, usually involves multiple vascular beds. Previous studies have shown that atherosclerotic disease that impacts both intracranial and extracranial territories is common in stroke patients [1-2]. By using angiographic techniques, investigators found that 43% of stroke patients developed concurrent arterial stenosis [2]. However, angiography only provides luminal information so may underestimate severity of atherosclerosis due to the phenomenon of positive remodeling [3]. Recently, 3D MR vessel wall imaging techniques, such as MERGE [4], SNAP [5], and VISTA [6], have been proposed for detection of high-risk plaque. Due to their large coverage, these 3D sequences have the potential to simultaneously capture concomitant high-risk plaques in intracranial and extracranial circulations. This study sought to assess concomitant extracranial and intracranial atherosclerosis in stroke patients using fast 3D multicontrast black-blood vessel wall imaging sequences. Effective detection of the concomitant high-risk plaques in multiple neurovascular beds can help ischemic stroke prevention.

**Methods:** Twenty-seven subjects (20 males. mean age 53.8 years) with recent ischemic stroke (≤ one week) underwent 3D multicontrast vessel wall imaging at a 3.0T whole body scanner (Achieva TX, Philips Medical System, Best, Netherlands) with a custom-designed 36-channel neurovascular coil. This coil yields high resolution, large coverage vessel wall imaging due to dedicated elements from aortic arch to the top of the head. ***MR imaging:*** Both extracranial and intracranial arteries were imaged using 3D multicontrast black-blood sequences including MERGE, SNAP, and VISTA with the following parameters: 3D MERGE: FFE, TR/TE 9.2/4.3 ms, flip angle 6° ; 3D SNAP: FFE, TR/TE 10/4.8 ms, flip angle 11° ; and 3D VISTA-T2W: TSE, TR/TE 2500/268 ms. All 3D sequences were acquired with the same FOV (250 mm x 160 mm x 40 mm) and isotropic spatial resolution (0.8 x 0.8 x 0.8 $mm^3$). The longitudinal coverage is 250 mm which sufficiently covers both intracranial and extracranial arterial structures without gaps. ***Data analysis:*** The 3D MERGE, SNAP and VISTA data were reconstructed using a Philips MR work station with MPR and curved approaches. The atherosclerotic plaque was defined as local wall thickening and determined at extracranial and intracranial arteries, including common carotid artery (CCA), internal carotid artery (C1-C7 [7]) middle cerebral artery (MCA), anterior cerebral artery (ACA), posterior cerebral artery (PCA), and basal artery (BA). The maximum wall thickness (MaxWT), length, and luminal stenosis for each lesion were measured. Presence or absence of lipid-rich necrotic core (LRNC) on VISTA-T2W (hypointense) and intraplaque hemorrhage (IPH) on SNAP (hyperintense) were respectively identified for each lesion. The concomitant plaques at intracranial (C2-C7, ACA, MCA, or BA) and extracranial (CCA or C1) arteries were evaluated.

**Results:** In total, 124 plaques were detected for all subjects. Of 27 subjects, 20 (74.1%) had concomitant intracranial and extracranial atherosclerotic disease, 6 (22.2%) and 1 (3.7%) had extracranial and intracranial plaques along, respectively. Compared to extracranial plaques, intracranial lesions exhibited significantly smaller MaxWT, length, and lower prevalence of IPH (Table 1). Intracranial lesions showed similar luminal stenosis and incidence of LRNC compared with extracranial arteries. Of 79 intracranial lesions, 28.9% had normal lumen size and 1/3 of lesions had mild luminal stenosis (Fig. 1). Similarly, 35.4% and 32.9% of 45 extracranial lesions appeared normal lumen and had mild stenosis, respectively (Fig. 1).

**Table 1. Comparison of atherosclerotic plaques**

| | Atherosclerotic plaque | | |
|---|---|---|---|
| | Intracranial arteries (N=45) | Extracranial arteries (N=79) | *P* |
| **MaxWT, mm** | 2.3±0.8 | 3.3±1.2 | <0.001 |
| **Length, mm** | 6.8±3.2 | 12.6±7.3 | <0.001 |
| **Stenosis, %** | 29.4±29 | 23.4±26.6 | 0.248 |
| **LRNC, N (%)** | 23 (51.1) | 45 (57) | 0.529 |
| **IPH, N (%)** | 4 (8.9) | 19 (24.1) | <0.001 |

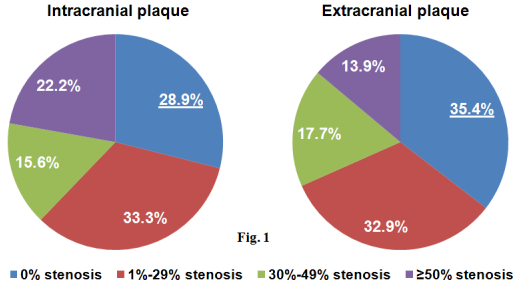


Fig. 1

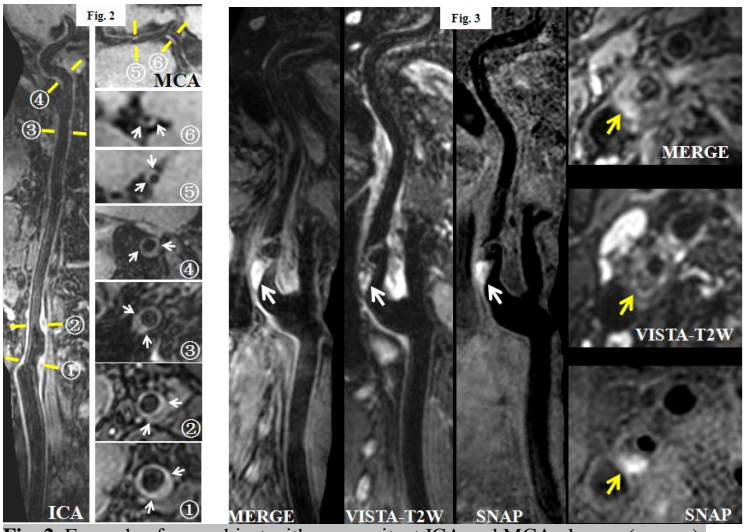


**Fig. 2.** Example of one subject with concomitant ICA and MCA plaques (arrows), ①~⑥ represent the cross-sectionally reconstructed images and their corresponding locations at curved reconstructed image.

**Fig. 3.** Multicontrast 3D images delineate a key ICA high-risk plaque feature: large LRNC (hypointense on VISTA-T2W) with IPH (hyperintense on SNAP).

**Discussion and Conclusions:** To the best of our knowledge, this is the first study to investigate concomitant intracranial and extracranial atherosclerotic disease using 3D MR black-blood vessel wall imaging. We found that concomitant intracranial and extracranial plaques are prevalent in stroke patients. In our study, the prevalence of concomitant plaque was significantly higher than literature reports (74.1% vs. 43% [2]). Fig. 2 is an example showing concomitant intracranial and extracranial plaques by 3D vessel wall imaging. Moreover, in our study, most intracranial and extracranial lesions show normal lumen size or mild stenosis, indicating that vessel wall imaging helps to detect more lesions compared to angiographic techniques. Benefiting from the large coverage (250 mm), isotropic high spatial resolution, and time efficiency, the 3D imaging sequences allow complete and comprehensive imaging of neurovascular atherosclerotic disease at multiple vascular beds without switching coils or imaging protocols during scans. More importantly, our multicontrast 3D imaging protocol enables fast comprehensive assessment of neurovascular plaque burden and compositional features including LRNC and IPH (Fig. 3) that are related to plaque vulnerability. This may optimize procedures for detection of culprit lesions in ischemic stroke subjects in emergency room settings.

**References:**

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