

Correlation of Atherosclerotic Disease between Extracranial and Intracranial Carotid Artery in Symptomatic Patients: A 3.0T MR Vessel Wall Imaging Study

Xu Han¹, Jianming Cai¹, Danqing Liu¹, Lin Ma¹, Youquan Cai¹, and Xihai Zhao²

¹Department of Radiology, PLA General Hospital, Beijing, China, ²Center for Biomedical Imaging Research & Department of Biomedical Engineering, Tsinghua University, Beijing, China

Introduction:

It has been shown that atherosclerotic disease is associated with ischemic cerebrovascular events [1-2]. It is well-established that atherosclerosis is systematic, ‘panvascular’ active inflammatory disease, involving multiple vascular beds. We hypothesized that characteristics of atherosclerotic plaque at one vascular bed might be an indicator for other vascular atherosclerosis. Previous studies have shown that the coexisting atherosclerotic disease at intracranial and extracranial vascular territories is common in stroke patients [3-4]. However, the correlation of atherosclerosis between intracranial and extracranial arteries has not been well studied.

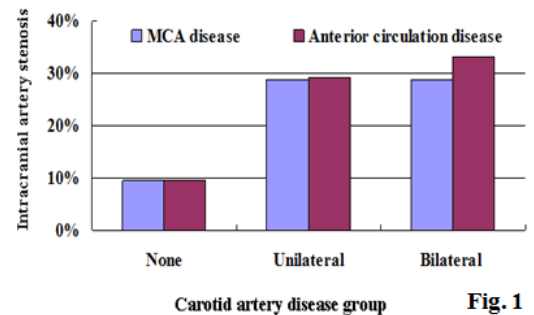
Purpose: This study sought to investigate the correlation of atherosclerotic disease between extracranial and intracranial carotid arteries in symptomatic patients.

Method:

One hundred and two patients with recent TIA or ischemic stroke underwent carotid black-blood vessel wall imaging and intracranial MR angiography (MRA) at a 3.0T whole body scanner (GE) with a 4-channel phase-array carotid coil and 8-channel head coil, respectively. **MR imaging:** Carotid vessel wall images were acquired using multicontrast sequences (T1w, PDw, T2w, and 3D-TOF) with the following parameters: T1w: quadruple inversion-recovery (QIR) [5], black-blood, 2D FSE, TR/TE 600/7.3ms; PDw and T2w: double IR, FSE, TR 3000 ms, TE 10 ms for PDw and 60 ms for T2w, echo train length 10; and 3D-TOF: SPGR, TR/TE 29/2.1ms, flip angle 20°. All axial images were acquired centered to the carotid bifurcation of the symptomatic arteries with field of view (FOV) of 14cm x 14cm² and acquisition matrix of 256 x 256. The slice thickness and longitudinal coverage were 2mm and 32mm for T1w and T2w imaging and 1mm and 40mm for 3D-TOF imaging, respectively. Intracranial arteries were imaged using 3D-TOF sequence with the following parameters: SPGR, TR/TE 18/2 ms, flip angle 15°, bandwidth 41.67, slice thickness 1mm, FOV 22 x 22cm², and acquisition matrix 448 x 224. **Data analysis:** The atherosclerotic lesions were measured at GE workstation (GE ADW 4.5) by one experienced radiologist. The maximum wall thickness, length, and luminal stenosis for each lesion were measured. In addition, the presence or absence of plaque compositions including calcification, LRNC, IPH and FCR was identified. The patients were divided into three groups: None group: no lesion at any carotid artery; unilateral group: the lesions occur on one side of carotid artery; and bilateral group: the lesions can be seen on bilateral carotid arteries. The incidence of each carotid plaque component was calculated for all the patients. The intracranial 3D-TOF images were reconstructed using maximum intensity project (MIP) algorithm to measure the luminal stenosis for intracranial lesions particularly for middle cerebral artery and anterior circulation arteries. The most severe intracranial stenosis was recorded for each patient. The intracranial stenosis was compared among patients in none, unilateral and bilateral groups.

Results:

Of 102 recruited patients, 52 had stroke and 50 had TIA. For extracranial carotid artery, MaxWT, length and luminal stenosis was 3.5±1.7mm, 27.7±15mm, and 36.4%±33.7%, respectively. Of 102 patients, 68 (66.7%) had calcification, 47 (46.1%) had LRNC, 46 (45.1%) had IPH, and 19 (18.6%) had FCR at carotid lesions. For intracranial artery disease, the luminal stenosis was 28.8%±26.8% for MCA and 29.3%±26.7% for anterior circulation arteries, respectively. The MCA stenosis was significantly correlated with carotid MaxWT ($r=0.426$, $P<0.001$) and luminal stenosis ($r=0.496$, $P<0.001$). Similarly, stenosis of anterior circulation arteries was significantly associated with carotid MaxWT ($r=0.447$, $P<0.001$) and luminal stenosis ($r=0.504$, $P<0.001$). There was no significant correlation between carotid plaque compositional features and intracranial artery disease. In addition, we found that severity of intracranial artery disease showed increasing trend with carotid artery disease involving none, unilateral and bilateral sides ($P<0.001$, Fig. 1).



Discussion and Conclusions:

This study investigated the correlation of atherosclerotic disease between intracranial and extracranial carotid arteries. The findings of significant correlation between intracranial and extracranial plaques further compel the evidence that atherosclerosis is a systemic disease that usually impacts multiple vascular territories. Fig. 2 is an example showing atherosclerotic plaques at both intracranial and extracranial arteries. We also found that there was significant correlation for severity of atherosclerotic disease between intracranial and extracranial circulations. Our findings suggest that atherosclerotic plaque at extracranial carotid artery might be an indicator for either presence or severity of intracranial atherosclerosis.

References:

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- [5] Yarnykh V, et al. *MRM*. 2002; 48:899-905.

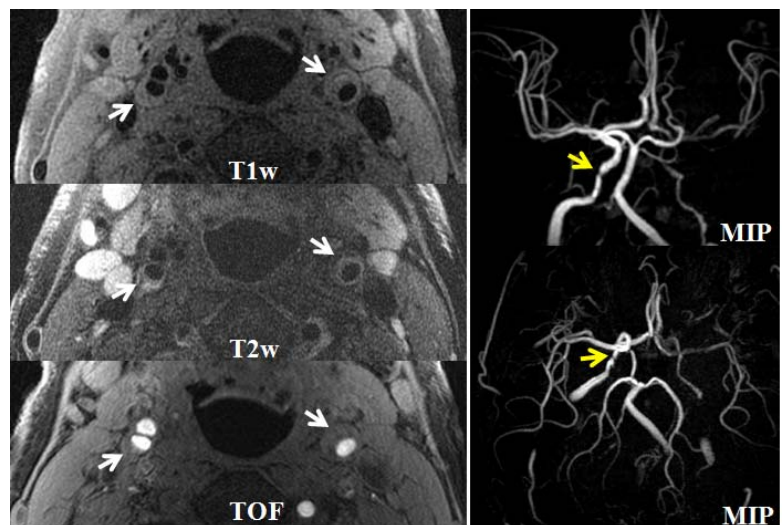


Fig. 2: atherosclerotic diseases can be seen at bilateral carotid arteries (white arrows) on multicontrast vessel wall images and right intracranial artery (yellow arrows). The left intracranial circulation showed diffuse lower blood perfusion due to the severe stenosis occurring at left internal carotid artery which is not showing in the left panel images.