An MRI Technique for the Study of the Prevalence of Atherosclerosis

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Introduction

Carotid artery wall thickness has been strongly correlated with systemic cardiovascular disease [1] and is a critical element for understanding local arterial remodeling [2]. High-resolution, black-blood MRI is a proven technique for the quantitative assessment of wall morphology in the common, bifurcation, and internal segments of the carotid artery [3]. Furthermore, measurements from carotid MRI have also been shown to be highly reproducible [4]. To date, however, protocols have focused on imaging only the artery with the more significant lesion as previously determined by duplex ultrasound. As such, an understanding of the occurrence of bilateral carotid disease remains undefined. We designed an MRI protocol, which optimized coverage of both arteries thereby enabling the study of bilateral carotid plaque burden. We applied this technique to a population of patients without a previous history of carotid disease to determine the prevalence of bilateral carotid atherosclerosis.

Methods

102 patients (56% female; 22% with diabetes; 51% smokers) without previous history of carotid disease underwent high-resolution carotid MRI on a 1.5T-scanner using T1 (TR/TE = 900/10.6 ms), T2 (TR/TE = 3700/42.7 ms), and PD-weighted (TR/TE = 3700/8.5 ms) sequences. Imaging parameters were FOV = 160 mm, matrix size = 256 x 256, and 2 averaged excitations. Axial images were acquired at 2 mm intervals for a total longitudinal coverage of 32 mm, centered at the bifurcation of the randomly assigned left or right carotid artery. Using a custom-designed image analysis tool (CASCADE, University of Washington) [5], two expert reviewers interpreted each scan by reaching a consensus opinion. For each artery, an AHA lesion score, maximum wall thickness (MAX), mean wall thickness (MWT), and the mean wall-to-outer-wall ratio (WOWR) were determined. Patients were identified as diseased if at least one slice on either side had an AHA score of 3 or higher. Measurements from each artery were compared to the contralateral side to determine the side with greater thickness. A paired student's T-test and Pearson's correlation coefficient was used to compare measurements from arteries with greater thickness to arteries with lesser thickness.

Results

Of the 102 patients, 14 patients (13.7%) were excluded due to poor image quality (n = 7) or failure to image the bifurcation (n = 7) on at least one side. Of the remaining patients, 81 (92.0%) were identified as having an AHA lesion score of 3 or higher in at least one artery and were categorized as diseased. Table 1 summarizes the results from comparing the artery of greater thickness to the artery of lesser thickness for all, diseased, and non-diseased patients. For all parameters there was a strong correlation between thickness measurements of the greater and lesser artery. Additionally, both the MAX and MWT of the greater vessel of diseased subjects were significantly greater than the respective measurements from the greater vessel of non-diseased subjects (p = 0.001; p = 0.003, respectively). Finally, measurements (Table 1, *italics*) of the lesser artery from diseased patients compared to measurements (Table 1, **bold**) of the greater artery from non-diseased patients were found to have a significantly thicker MAX (p = 0.016) and MWT (p = 0.049).

Conclusion

This study represents the first MRI comparison of bilateral carotid artery wall measurements in patients without a previous history of carotid disease. The new MRI protocol enabled adequate coverage of both carotid arteries in a vast majority of the patients. Consequently, we found that patients with carotid disease on one side had increased thickness of the contralateral artery (Fig. 1), which was significantly greater than a normal artery. These findings illustrate the feasibility and the importance of following carotid disease bilaterally.

Table 1. Mean values for each wall measurement. The P value is derived from a paired student's T-test comparing the "Greater" column to the "Lesser" column.

All (n = 88)	Greater	Lesser	P	Pearson's R
MAX	2.528 ± 0.932	1.956 ± 0.554	0.001	0.628
MWT	1.160 ± 0.166	1.073± 0.130	0.001	0.850
WOWR	0.444±0.054	0.415± 0.045	0.001	0.858
Diseased (n = 81)				
MAX	2.595± 0.942	*1.991± 0.561*	0.001	0.607
MWT	1.172± 0.167	*1.079± 0.132*	0.001	0.845
WOWR	0.444± 0.056	0.415± 0.046	0.001	0.860
Non-diseased (n = 7)				
MAX	**1.761 ± 0.164**	1.543 ± 0.230	0.004	0.766
MWT	**1.022 ± 0.072**	1.002 ± 0.082	0.002	0.997
WOWR	0.439 ± 0.024	0.413 ± 0.028	0.003	0.817

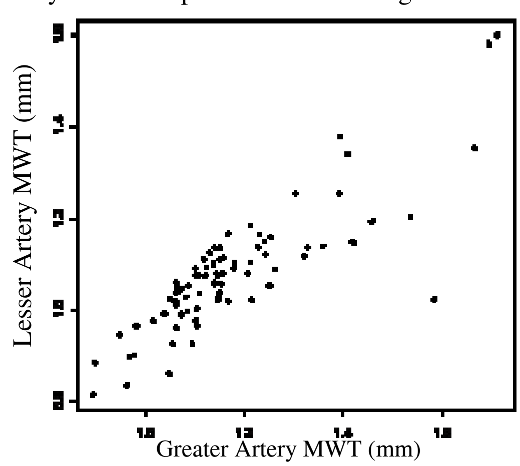

Figure 1. Plot of "Greater" MWT vs "Lesser" MWT for diseased subjects. Pearson's correlation coefficient was R = 0.845.

References

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