Distribution of AHA Type VI Atherosclerotic Lesions in the Carotid Artery and Relationship to Quantitative Plaque Measurements: An in vivo MRI Study

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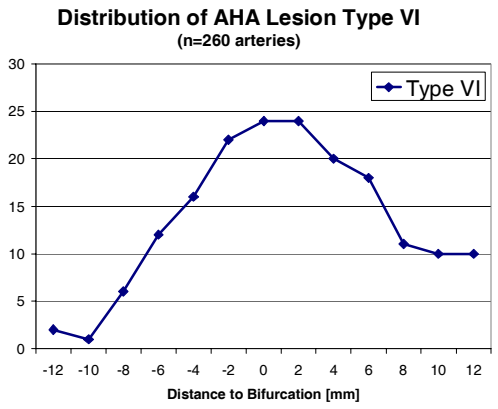
Introduction: Arterial stenosis alone has been shown to be a poor predictor of cardiovascular events, and it is commonly believed that AHA Type VI lesions (AHA-LT6, lesions with luminal surface defect, hemorrhage/thrombus or calcified nodule) are potentially culprit lesions which affect a plaque's vulnerability (1). However, little is known about the prevalence of AHA-LT6 in different segments of the arterial wall and its relationship to lumen, wall and outer wall dimensions. MRI is able to accurately and reproducibly measure carotid atherosclerotic lumen and wall dimensions (2) and to classify atherosclerotic lesion according to a modified American Heart Association (AHA) classification scheme (3). Via magnetic resonance imaging (MRI), we sought (i.) to determine the prevalence of AHA-LT6 in different segments of the carotid artery and (ii.) to compare quantitative plaque measurements of axial locations with and without AHA-LT6.

Methods: 175 subjects with varying degrees of stenosis as measured by duplex ultrasound (1-99%) had their bilateral carotid arteries imaged at 1.5T using a multi-sequence protocol. After excluding arteries with a previous endarterectomy, poor image quality, or missing ultrasound data, there were 260 arteries with 2504 axial MR image locations available for analysis. 2 Expert readers, measured the lumen (LA), wall (WA) and total vessel areas (TVA=WA + LA) and determined the AHA –LT6 on the cross-sectional images in consensus decision. The presence of AHA-LT6 was based on the relative tissue intensities in T1-, T2-, PD- and TOF-weighted images and the appearance of the luminal surface (3). The prevalence of AHA-LT6 was analyzed for each MR imaging location relative to the bifurcation and for each segment of the artery. Arterial segments were defined as: 1) common: >4 mm proximal to the bifurcation 2) carotid bulb: 0-4 mm proximal to the bifurcation and 3) internal: ≥ 2 mm distal to the bifurcation. To compare the prevalence of AHA-LT6 between the common and internal carotid artery and the carotid bulb and to compare quantitative measurements between locations with and without AHA-LT6, a paired t-test was used.

Results (see table 1 and figure):

Axial locations with AHA-LT6 had a significantly smaller mean LA (22.9 vs. 36.1 mm^2; p<0.001) and a larger mean WA (59.4 vs. 44.3 mm^2; p=0.001) compared to locations without AHA-LT6. Mean TVA did not differ significantly between locations with and without AHA-LT6 (82.4 vs. 80.3 mm^2; p=0.3). Although mean LA was significantly higher in the carotid bulb compared to the internal carotid artery (44.6 mm^2 vs. 24.4 mm^2; p<0.001) and common carotid artery (44.6 mm^2 vs. 39.6 mm^2; p<0.001), AHA-LT6 had a significantly higher prevalence in the carotid bulb compared to the common (21.6 vs. 6.6%; p<0.001) and internal carotid artery (21.6 vs. 16.7%; p=0.01). AHA-LT6 was more common in the internal than in the common carotid artery (p<0.001). Mean WA was higher in the carotid bulb compared to the internal (61.1 mm^2 vs. 38.9 mm^2; p<0.001) and common carotid artery (61.1 mm^2 vs. 41.1 mm^2; p<0.001). The figure demonstrates the prevalence of complicated AHA Type VI lesions by MR imaging locations with the x-axis indicating the distance from the bifurcation (2 mm steps).



Conclusion:

This study showed that locations with AHA-LT6 tend to have a smaller LA compared to locations without AHA-LT6. However, the highest prevalence of AHA-LT6 was found in the carotid bulb, the arterial segment with the largest mean LA and mean WA. These findings indicate that other factors besides luminal stenosis, such as mean WA as an indicator of plaque burden and geometrical factors, such as the location of a lesion within the arterial tree, determine a plaque's vulnerability.

References

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