Association between carotid plaque characteristics and cerebral white matter lesions

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Purpose. To prospectively assess the relation between carotid plaque characteristics and the development of new cerebral white matter lesions (WMLs) at MRI. It is known that there is a relation between carotid atherosclerosis and WMLs but it is unclear yet whether this relation indirect via shared risk factors or causative in nature.

Methods. Fifty TIA/stroke patients with ipsilateral 30-69% carotid stenosis underwent MRI of the plaque and brain at baseline. Brain MRI was repeated after one year. The protocol for carotid plaque MRI consisted of T1-weighted turbo field-echo (TFE), time-of-flight (TOF), T2-weighted turbo spin-echo (TSE), and pre- and post-gadopentetate dimeglumine-enhanced T1-weighted TSE images. The protocol for brain MRI consisted of T2-weighted TSE and fluid-attenuated inversion recovery (FLAIR) TSE images. For each plaque, lipid-rich necrotic core (LRNC) volume, fibrous cap (FC) status and intraplaque hemorrhage (IPH) were assessed (Figure 1). Cerebral WMLs (Figure 2) were quantified with a semiautomatic method.

Results. Mean WML volume significantly increased over a one-year period (6.52±1.16 [SE] vs. 6.97±1.26 ml, P=0.005). Age and hypertension were significantly associated with baseline WML volume (Spearman ρ=0.542, P<0.001; and Spearman ρ=0.329, P=0.020, respectively). Age and baseline WML volume were significantly associated with WML progression (Spearman ρ=0.371, P=0.008; and Spearman ρ=0.483, P<0.001, respectively). Baseline WML volume and WML progression did not significantly differ between patients with 30-49% and 50-69% stenosis (P=0.689 and P=0.342, respectively). There were no significant associations between LRNC volume and baseline WML volume (Spearman ρ=0.088, P=0.545) and WML progression (Spearman ρ=0.053, P=0.715). Baseline WML volume and WML progression did not significantly differ between patients with a thick and intact FC and patients with a thin and/or ruptured FC (P=0.504 and P=0.867, respectively), and also not between patients with and without IPH (P=0.700 and P=0.917, respectively).

Conclusions. In TIA/stroke patients with carotid stenosis, we found no associations between carotid plaque characteristics and ipsilateral WML severity and progression over a one-year period. This suggests that there is no causal relationship between carotid plaque vulnerability and the occurrence of WMLs.

Figure 1. Co-registered T1w TFE, TOF, T2w TSE, pre- and post-contrast T1w TSE images of a transverse section of a carotid plaque. The right bottom panel displays the different plaque components: red=lumen; green=outer vessel wall; yellow=LRNC; orange=calcifications; remaining vessel wall area=fibrous tissue. IPH was scored as being present (asterisks in T1w TFE and TOF images) and the FC was designated as thin and/or ruptured (arrow in post-contrast T1w TSE image).

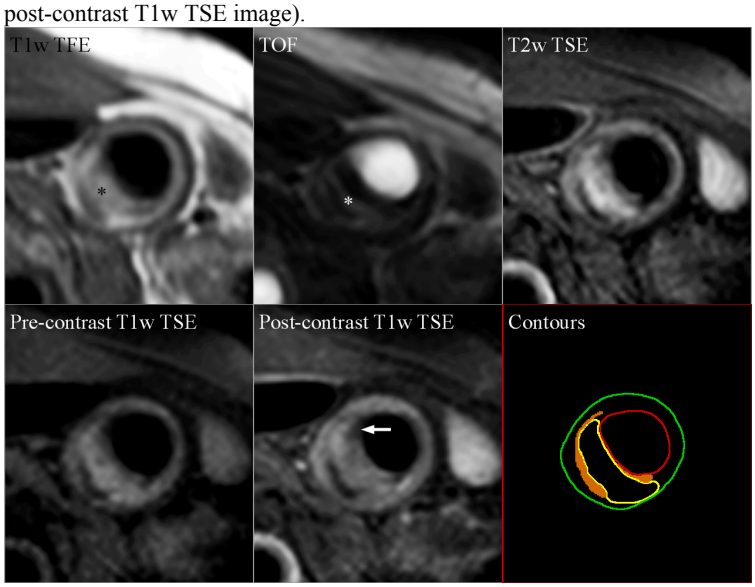
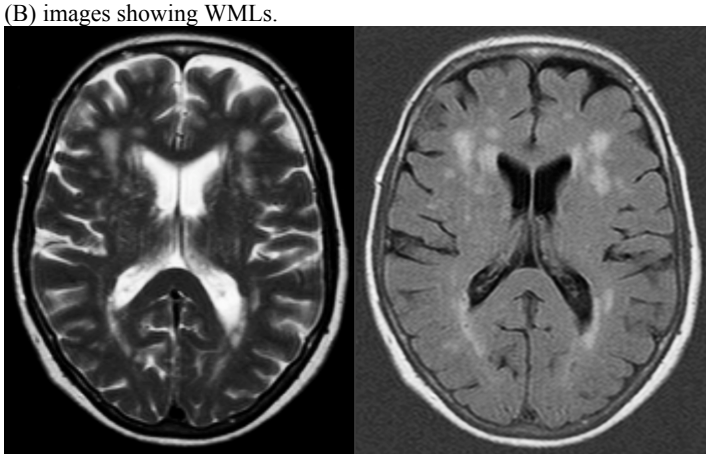


Figure 2. WMLs identified at co-registered T2w TSE (A) and FLAIR (B) images showing WMLs.



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