

Comparison of plaque imaging and luminal stenosis to discriminate clinical presentation in middle cerebral artery disease

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Introduction: Intracranial atherosclerosis (IA) is a major substrate for stroke, accounting for 5-10% of strokes in western societies^{1,2} and 33-55% of strokes^{3,4} in Asian populations. IA is known to affect the middle cerebral artery (MCA), as well as intracranial segments of the internal carotid, vertebrobasilar, posterior, and anterior cerebral arteries. Overall, the MCA accounts for approximately 40% of IA due to its dominance and anatomical location⁵⁻⁷. Despite of the serious disease burden of IA, few studies have concentrated on this disorder, instead focusing on extracranial carotid atherosclerosis, as this is more common to western societies and may be amenable to surgical intervention. Accordingly, relatively little is known about the mechanism for stroke in intrinsic MCA stenosis and there is an absence of clinical criteria for assessing intracranial disease severity.

Aim: To assess the complementary value of MCA wall imaging to luminal stenosis in differentiating patient clinical presentation.

Methods: (1) **MRI acquisition** One hundred and eleven patients underwent high-resolution black-blood MR imaging of their MCAs (**Figure 1**). Eighty eight patients were symptomatic and the other twenty three were asymptomatic. Time of flight (TOF), T1, T2-weighted and contrast-enhanced T1-weighted images covering the entire MCA stenosis were acquired. Image matrix was 256x256 and field of view 10x10 cm²; (2) **Data analysis** Luminal stenosis was computed at the most stenotic site, referring to the proximal healthy section. Manual segmentation of plaque outer wall and lumen was performed to compute plaque burden (PB). T1 signal ratio was computed by normalizing the wall signal intensity in the T1 weighted image to the value of nearby grey matter and the enhancement index was computed by the difference of this ratio in pre- and post-contrast images.

Results: Both PB and luminal stenosis of culprit lesions were significantly higher than those of asymptomatic lesions (PB: 86.2%±13.8% vs. 75.4%±17.1%, $p=0.001$; Stenosis: 63.6%±24.1% vs. 55.6%±24.8%, $p=0.005$). High signal intensity in T1-weighted image may imply hemorrhage-like content, which has been observed to be a risk factor for plaque vulnerability in the carotid artery. However, in this study there is no significant difference in term of the T1 signal ratio between culprit and asymptomatic lesions (1.02 ± 0.25 vs. 1.10 ± 0.46 , $p=0.34$). In contrast, the enhancement index in culprit lesions was significant bigger than asymptomatic lesions (0.66 ± 0.34 vs. 0.13 ± 0.08 , $p=0.004$). These findings imply that plaque inflammation and neovascularization are more prevalent in culprit when compared with asymptomatic lesions. Receiver operating characteristic analysis (**Figure 2**) indicated that the predictive accuracy of PB to discriminate clinical presentation was higher than luminal stenosis (PB 0.70 95% CI [0.61, 0.79], $p<0.001$ vs. Stenosis 0.63 [0.54, 0.73], $p<0.001$), however the improvement seen was not statistically significant ($p=0.37$). The optimal PB value to identify a culprit plaque was 71.5% (69.3% were culprit lesions when PB was higher than this value, while 73.5% of lesions were asymptomatic if PB was lower). For stenosis this value was 61.8% (70.5% were culprit lesions when stenosis was higher than this value, while 50% of lesions were asymptomatic when stenosis was lower than this value).

Conclusions: High-resolution MCA plaque wall imaging using 2D Turbo MR sequences provides limited additional value to luminal stenosis. More advanced MR technologies and additional analyses, including mechanical simulations modelling plaque stress, are required to reliably discriminate patient clinical presentation.

References:

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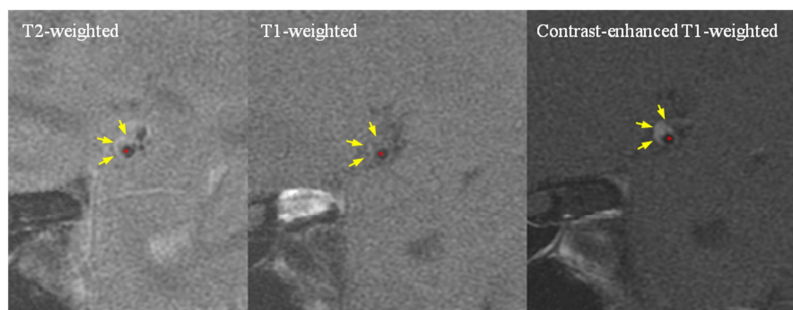


Figure 1. MR images showing a plaque in the middle cerebral artery of a 35-year old, male, symptomatic patient.

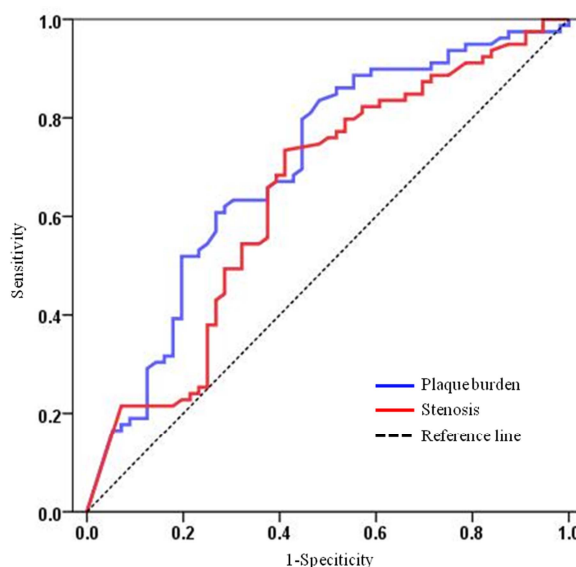


Figure 2. Receiver operating characteristic (ROC) curves illustrating predictive abilities of both plaque burden and stenosis