

Combined MRI and TCD to assess association between different vulnerable plaque features in stroke patients

Martine Truijman^{1,2}, Alexandra de Rotte³, Rune Aaslid⁴, Anouk van Dijk^{5,6}, Madike Liem⁷, Floris Schreuder^{2,8}, Robert van Oostenbrugge⁸, Joachim Wildberger¹, Paul Nederkoorn⁷, Jeroen Hendrikse³, Aad van der Lugt⁵, Werner Mess², and Marianne Eline Kooi¹

¹Radiology, Maastricht University Medical Center, Maastricht, Netherlands, ²Clinical Neurophysiology, Maastricht University Medical Center, Maastricht, Netherlands, ³Radiology, University Medical Center, Utrecht, Netherlands, ⁴Hemodynamics AG, Bern, Switzerland, ⁵Radiology, Erasmus Medical Center, Rotterdam, Netherlands, ⁶Neurology, Erasmus Medical Center, Rotterdam, Netherlands, ⁷Neurology, Academic Medical Center, Amsterdam, Netherlands, ⁸Neurology, Maastricht University Medical Center, Maastricht, Netherlands

Target audience: Researchers involved in imaging of carotid atherosclerotic plaques

Purpose: Being able to identify vulnerable atherosclerotic plaques in patients with a mild to moderate (30-69%) symptomatic carotid artery stenosis would highly contribute to clinical decision making. Intraplaque haemorrhage (IPH) and fibrous cap status (FC) are associated with an increased risk of a recurrent stroke⁽¹⁾. Both features can be well assessed with magnetic resonance imaging (MRI). In addition, the presence of microembolic signals (MES) are also associated with an increased risk of a recurrent stroke⁽²⁾. These signals can be determined with transcranial Doppler ultrasound (TCD). The main objective of this study was to investigate whether there is a relationship between the presence of MES detected with TCD and the presence of IPH and thin/ruptured FC on MRI.

Methods: Patients from four different academic and their surrounding regional hospitals with a transient ischemic attack (TIA) or minor stroke in the carotid territory and ipsilateral carotid plaque causing a mild to moderate stenosis were eligible for inclusion⁽³⁾. Exclusion criteria were a possible other cause of embolism (e.g. atrial fibrillation) or a renal clearance <30 ml/min. All patients underwent a multisequence MRI protocol on a 3T whole body scanner using a dedicated eight-channel phased-array coil (Shanghai Chenguang Medical Technologies Co., Shanghai, China) or a dedicated four-channel carotid phased-array coil with an angulated setup (Machnet B.V., Roden, the Netherlands). T1w pre- and postcontrast QIR TSE or T1w DIR FSE images were obtained as well as T1w IR-TFE or SPGR as previously described⁽³⁾. The post-contrast images were acquired 6 minutes after injection of 0.1 mmol/kg body weight of a gadolinium-based contrast. IPH was scored as being present if a hyperintense signal (compared with the adjacent sternocleidomastoid muscle) was visible in the bulk of the plaque on IR-TFE or SPGR images (figure 1). FC status was scored by comparing pre- and postcontrast images for the presence of a continuous high signal between LRNC and the lumen (thick FC) or an interrupted or no continuous hyperintense signal (thin/ruptured FC) (figure 1). TCD measurements were performed on the symptomatic side with an ambulatory TCD system (TCD-X, Hemodynamics AG, Bern, Switzerland) for the duration of four hours. Analysis of the data was done with semi-automatic detection software, which has been validated by 2 independent human observers. A Fisher's exact test was performed to determine whether there is a significant difference in MES in patients with and without IPH or a thin/ruptured FC.

Results: In total 113 patients (88 male) were included, with a mean age of 67 (39-81) years. 8 patients were excluded due to too much noise artifacts during TCD recording, leading to 105 eligible patients. Due to the absence (n=7) and poor image quality (n=6) of the postcontrast images, in 92 patients the FC status was analyzed. The mean number of days was 50 (11-100) and 51 (12-100) between event and TCD and MRI, respectively. In 44/105 (41.9%) of the patients, IPH was present and 36/92 (39.1%) patients had a thin/ruptured FC. 44/92 patients (48%) had neither IPH nor a thin/ruptured fibrous cap. In total 23034 minutes of TCD signal were recorded, with a mean of 219 minutes per patient (21-379). 28 MES were detected in 8/105 (7.6%) patients. 6/8 patients with MES had no IPH on MRI and 6/8 different patients with MES had no thin/ruptured FC. The Fisher's exact test revealed no significant difference in MES between patients with and without IPH ($p = 0.46$) or thin/ruptured FC ($p = 0.48$). There was also no significant difference in MES between patients who had no vulnerable plaque features on MRI i.e. patients who had either IPH and/or a thin/ruptured FC ($p=0.47$).

Discussion and Conclusion: Patients with IPH or thin/ruptured FC do not have significantly more MES. IPH and FC status examined with MRI are not related with MES detected by TCD and thus MRI and TCD provide additive information on plaque vulnerability.

Acknowledgements: This research was performed within the framework of CTMM, project PARISK (grant 01C-202), and supported by the Netherlands Heart Foundation.

References: 1) Gupta et al., Stroke. 2013;44:3071; 2) Ritter et al., J Neurol. 2008;255:953; 3) Truijman et al., Int J Stroke, epub 21 Oct 2013

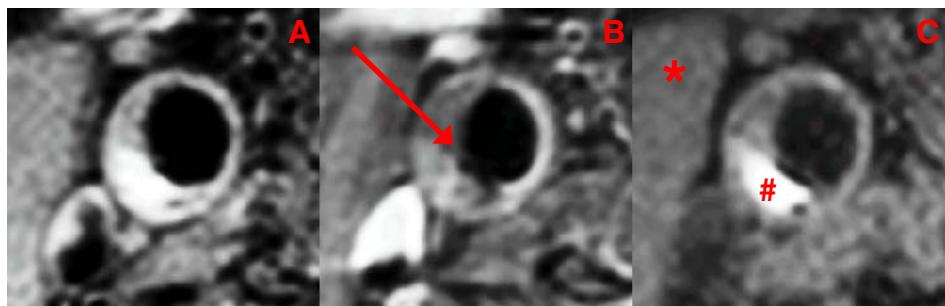


Figure 1: MR images showing T1w QIR TSE pre- (A) and postcontrast (B) and T1w IR-TFE (C). Note the absence of a continuous high signal between LRNC and lumen (arrow) indicating a thin or ruptured fibrous cap and the hyperintense signal (#) in the IR-TFE image compared with the sternocleidomastoid muscle (*) indicating IPH.