# Diagnostic Impact of Aortic MRI at 3Tesla in Acute Stroke Patients

A. Harloff[1], S. Brendecke[1], J. Simon[1], W. Wallis[1], D. Assefa[2], T. Helbing[2], A. Frydrychowicz[3], J. Weber[4], C. Weiller[1], J. Hennig[3], and M. Markl[3]

[1]Neurology, University Hospital Freiburg, Freiburg, Germany, [2]Cardiology and Angiology, University Hospital Freiburg, Freiburg, Germany, [3]Diagnostic Radiology, MR Physics, University Hospital Freiburg, Freiburg, Germany, [4]Neuroradiology, University Hospital Freiburg, Freiburg, Germany

**Introduction:** Complex aortic plaques (≥4 mm thickness or aortic thrombi, or both) are considered a major cause for brain ischemia and constitute the most frequent high-risk pathology detected by the gold standard transesophageal echocardiography (TEE) [1, 2]. Drawbacks of TEE are related to its semi-invasiveness and limitations due to air artifacts and insonation angles. Therefore, assessment of plaques especially in the proximal ascending aorta and aortic arch, the origin of the brain supplying great arteries, is often not possible. In contrast, multi-slice two-dimensional (2D) or true three-dimensional (3D) MRI offers complete coverage of the entire aorta [3]. In a previous study with 73 patients we were able to compensate TEE limitations and demonstrated that MRI was superior to TEE for the detection of aortic high-risk plaques [4]. However, this study included non-consequent patients with acute stroke with heterogeneous aortic atherosclerosis ranging from normal aortic wall to severe aortic atherosclerosis. Moreover, the previous MR protocol did not include multi-contrast evaluation of detected plaques. Thus, in an ongoing follow up study we consecutively included only acute stroke patients with advanced stages of aortic atherosclerosis as confirmed by in TEE. Moreover, multi-contrast T1, T2, and PD weighted and 3D CINE imaging were performed.

**Methods:** To date, 68 consecutive acute stroke patients admitted to our hospital were prospectively included. With a current recruitment rate of 16 – 20 patients/month ~150 patients are expected to be included until March 2009. Inclusion criteria were: acute cerebral and/or retinal ischemia, age ≥18 years, undetermined stroke etiology prior to TEE, aortic plaque ≥3 mm in TEE, no contraindication against MRI at 3 Tesla.
MRI was performed on a 3T routine system using an eight-channel body coil (TRIO, Siemens, Erlangen, Germany). In patients suitable for contrast agent application time-resolved contrast enhanced 3D MR angiography (tr-CE-MRA) was performed. Spatial acceleration (GRAPPA, R = 4) and view sharing along the temporal direction (TREAT) resulted in a reconstructed voxel size of 1.0x1.3x1.2 $mm^3$ and effective temporal update rate of 3.8s. Next, ECG-gated 2D CINE gradient echo imaging normal to the ascending and descending aorta was performed to individually determine the temporal window within the cardiac cycle with least vessel motion. Subsequent ECG gated high spatial resolution plaque imaging was performed using a T1-weighted rf-spoiled and fat saturated 3D gradient echo sequence covering the entire throracic aorta (acquisition window = 157ms, individually adjusted ECG delay, TE / TR = 2.3ms / 5.5ms, flip angel = $20^o$, GRAPPA with R = 2, spatial resolution 0.9x1.1x1.1$mm^3$). To permit data acquisition during free breathing and to minimize artifacts respiration control based on dynamically adapted navigator gating with respiration drift correction was implemented [5]. In the presence of aortic plaques ≥4 mm additional multi-slice T2 (TE = 78ms) weighted 2D TSE imaging and optional multi-slice PD (TE = 12ms) weighted 2D TSE imaging (ECG gated, diastolic acquisition window, TR = 2RR intervals, flip angel = $90^o$-$180^o$, spatial resolution 1.1x1.2x3$mm^3$) at the site of aortic plaques were applied. In plaques with eccentric protrusion into the lumen and thickness ≥4 mm 3D CINE T1 sequences (TE / TR = 1.8ms / 3.3ms, flip angel = $15^o$, spatial resolution 1.3x1.5x1.3$mm^3$, temporal resolution = 53ms) were performed in order to screen for aortic thrombi.
The number of complex aortic plaques (≥4 mm thickness or aortic thrombi, or both) detected by TEE and MRI in the thoracic aorta were compared. Readers of TEE and MRI were blinded to patients` demographics and to results of the complementary imaging modality. Image quality in TEE and MRI for each aortic segment was graded as: 1 = low, 2 = moderate and 3 = good.

**Results:** Of 68 patients included in the study, 7 had to be removed from analysis due to the following reasons: TEE could not be performed: n = 1, diagnosis on discharge from hospital was another than cerebral or retinal ischemia: n = 2, MRI image quality was insufficient for reliable image reading: n = 4. Accordingly, 61 patients (mean age 66.0±9.3, 21 females) were available for final analysis. Exemplary image quality achieved with the MRI imaging protocol is shown for one patient in Figure 1. Table 1 summarizes image quality for both techniques. While lowest image quality in TEE was found for the aortic arch, assessment of the ascending and descending aorta was moderate to good in almost all patients. In contrast, MRI provided similar visualization quality (moderate in 1/3, good in 2/3 of the patients) for all aortic segments. The frequency of complex plaques detected by TEE versus MRI is given in Table 2. The number of complex plaques increased from the ascending to the descending aorta and was higher in MRI compared to TEE. Particularly, the number of plaques detected by MRI in the aortic arch was more than three times higher compared to TEE.

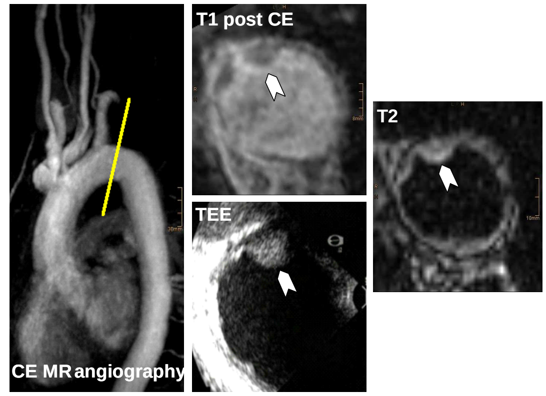


**Figure 1**: MR angiography indicates plaque location in the proximal descending aorta. A large protruding plaque is clearly visible as hypointense structure in T1 and T2 weighted imaging and is consistent with plaque morphology as shown by TEE (white arrows).

| Image grading | Asc. aorta TEE | Asc. aorta MRI | Aortic arch TEE | Aortic arch MRI | Desc. aorta TEE | Desc. aorta MRI |
|---|---|---|---|---|---|---|
| 1 - low | 0 | 0 | 49 | 0 | 0 | 0 |
| 2 - mod. | 48 | 15 | 6 | 25 | 1 | 18 |
| 3 - good | 1 | 46 | 5 | 36 | 60 | 43 |

**Table 1.** Grading of image quality in the three aortic segments in TEE versus MRI.

| Segment | Complex aortic plaques TEE | Complex aortic plaques MRI |
|---|---|---|
| Ascending aorta - n | 3 | 6 |
| Aortic arch - n | 5 | 18 |
| Descending aorta- n | 30 | 58 |
| All - n | 38 | 82 |

**Table 2.** Number of complex plaques detected by TEE versus MRI in each aortic segment of all 61 patients.

**Discussion:** To date, 61 acute consecutive stroke patients were successfully examined in this ongoing study. The increase of plaque frequency from the ascending to the descending aorta is in line with findings in large TEE studies [6]. Consistent with our previous MRI study [2], the number of complex plaque detected by MRI was significantly higher than in TEE, particularly for the aortic arch. This is most probable due to the superior visualization of the ascending aorta and the aortic arch by MRI which provides a three-dimensional coverage of the entire aorta and is not limited by insonation angles and air artefacts hampering assessment by TEE. We successfully performed additional T2 2D multi-slice imaging and optional PD weighted imaging. Thus, in contrast to our previous study [2] and by adding multi-contrast imaging we were able to further improve reliability for plaque identification and to discriminate wall thickening from insufficient blood suppression in T1 weighted imaging mimicking aortic plaques. In addition, 3D T1 CINE imaging allowed for the differentiation of aortic thrombi. Our findings indicate that MRI is more reliable for the detection of complex plaques within the entire thoracic aorta compared to TEE. Thus, 3D MRI it is a valuable imaging tool for patients with ischemic stroke, particularly for those with undetermined etiology despite complete diagnostic workup including TEE.

**Acknowledgement:** Grant Sponsors: DFG, Grant # MA 2383/4-1 and by the Bundesministerium für Bildung und Forschung (BMBF), Grant # 01EV0706.

**References:** [1] Reynolds HR et al. Curr Opin Cardiol. 2003;18:340-345. [2] Harloff A et al. Stroke. 2006;37:859-864. [3] Fayad ZA et al. Circulation. 2000;101:2503-2509. [4] Harloff A et al. J Neurol Neurosurg Psychiatry. 2008;79:540-6. [5] Markl M J Magn Reson Imaging. 2007;25:824-31. [6] Amarenco P et al. N Engl J Med. 1994;331:1474-9.