Purpose: Spontaneous cervical artery dissection (CAD) causes up to 20% of strokes among young patients. A quick and reliable diagnosis is of paramount importance to start immediate anticoagulant therapy. MRI has become the method of choice in the first line evaluation of patients with suspected CAD due to its capacity to directly demonstrate an intramural hematoma and an increased vessel diameter. However, reliable identification of acute CAD might be impaired by the limited spatial resolution of standard 1.5T MRI. In this prospective study we evaluated the diagnostic capacity of a high-resolution arterial vessel-wall-imaging protocol at 3.0T for the assessment of the complex pathomorphology of the vessel wall in CAD. Furthermore, findings at 3.0T were compared to 1.5T standard MRI.

Methods and materials: 38 consecutive patients with clinical signs of CAD (20 m, 16 f, mean age 41 yrs) underwent cervical MRI at 3.0T. MRI was acquired using a head-neck coil. The protocol consisted of fat suppressed axial T1w-TSE pre and post Gadolinium (TR/TE /ETL= 550 ms/8ms/3, 40 slices, reconstructed voxel size 0.45x0.45x4.0mm), axial T2w-TSE (TR/TE/ETL= 4000ms/80ms/21, 40 slices, 0.45x0.45x4.0mm) and CE-MRA (TR/TE/FA= 4.5ms/1.6ms/40°, 120 slices, 0.59x0.59x0.8mm). 3.0T-MRI was acquired using a phased array coil with four circular elements. The protocol consisted of bright blood 3D inflow MRA (TR/TE/FA = 25ms/3.1ms/16°, 120 slices, reconstructed voxel size 0.3x0.3x0.8mm); axial black blood cardiac gated water selective T1w-3D-FFE (TR/TE/FA = 31ms/7.7ms/15°, 36 slices, 0.3x0.3x1.0 mm) and axial black blood cardiac triggered fat suppressed T2w-TSE (TR/TE/ETL= 336ms/44ms/7, 18 slices, 0.3x0.3x2mm).

Three radiologists in consensus assessed both studies separately. At 3.0T special care was taken to identify the luminal and adventitial vessel boundary, the presence of a communicating intimal tear and the extent and visibility of any intramural hematoma (Fig.1). For both field strengths the presence of specific (mural hematoma, increased external diameter, pseudoaneurysm, string sign) and unspecific signs (occlusion, stenosis, hyperintense lumen, perivascular enhancement) was noted. CAD was judged definite, if any one specific sign was present, undetermined if an unspecific sign and normal if no pathologic signs were present. MR-diagnosis was verified by clinical and radiological follow-up.

Results: 3.0T-MRI provided excellent delineation of the vessel wall, due to nearly complete suppression of arterial blood signal (Fig. 2). In 17 patients CAD could be ruled out by both exams, in 13 patients dissection diagnosed at 1.5T was confirmed by 3.0T. In 2 patients findings were normal at 1.5T, whereas a dissection could be demonstrated at 3.0T. In 6 patients no conclusive findings were available at 1.5T; in 4 of these patients CAD could be demonstrated at 3.0T, whereas in 2 patients findings were normal. An intramural hematoma could be detected at 3.0T in 17 patients vs 9 patients at 1.5T. In 13/17 patients with confirmed CAD at 3.0T luminal and adventitial vessel wall boundary confining the intramural hematoma could be identified. In no patient an intimal tear was visible. An increased external diameter was detected in 14 patients at 3.0T vs 6 patients at 1.5T.

Conclusion: 3.0-MRI permitted excellent analysis of the morphological features of CAD due to increased spatial resolution. Detection rates of intramural hematoma and vessel wall expansion improved accordingly, permitting the definite diagnosis of CAD in approximately 30% more patients as compared to 1.5T-MRI. Thus, high resolution MRI at 3.0T not only increases the diagnostic performance of MRI for the diagnosis of CAD, but may also give new insights into the underlying complex pathomorphology.