MRI-Based Separation of Congenital and Acquired Vertebrobasilar Artery Anomalies in Ischemic Stroke of the Posterior Circulation

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Background and Purpose—Stroke MRI protocols provide useful information about underlying vessel pathologies in the anterior circulation by means of intracranial time-of-flight angiography. However, these protocols mostly fail in the posterior circulation to differentiate between congenital variants and secondary thrombosis. Therefore, a high-resolution anatomic True Fast Imaging in Steady State Precession sequence, added to a commonly used stroke imaging protocol, was evaluated.

Methods—MRIs of all emergency admissions to the stroke unit over 2 months were analyzed. Variations in the posterior circulation as displayed by time-of-flight and by the True Fast Imaging in Steady State Precession sequence, respectively, were graded by 2 readers blinded to the diagnosis.

Results—In the time-of-flight angiography, 50% of patients presented with distinctive vertebrobasilar alterations. Half of these were judged as high-grade anomalies, of which the True Fast Imaging in Steady State Precession sequence identified 25% as hypoplasia. In 40% of all patients with posterior ischemia, the True Fast Imaging in Steady State Precession sequence confirmed an acquired occlusion of the vertebrobasilar arteries.

Conclusions—The addition of an anatomic (True Fast Imaging in Steady State Precession) to a functional sequence (time-of-flight) in stroke MRI protocols enables the differentiation between artery occlusions and hypoplastic variants of the vertebral arteries. (Stroke. 2008;39:2382-2384.)

Key Words: anatomy ■ clinical neurology ■ imaging ■ MRI ■ vertebrobasilar disease

Standard angiographic imaging techniques like time-of-flight (TOF) magnetic resonance angiography (MRA) often fail to differentiate between occlusions and hypoplasia/aplasia of the vertebrobasilar vessels. The present study therefore addressed the potential of a structural MR sequence added to a routine stroke MRI protocol to reveal acquired thrombosis or stenosis by identifying the congenital anatomy of the vertebrobasilar system.

Materials and Methods

Study Cohort
In a retrospective study over 2 months, all emergency admissions to the Department of Neurology, University of Ulm were evaluated. Exclusion criteria were incomplete imaging data.

Imaging
All imaging was performed on a 1.5-T MRI Scanner (Siemens Symphony, Erlangen, Germany). The routine MRI protocol, including TOF angiography, was supplemented by a coronally oriented 3-dimensional True Fast Imaging in Steady State Precession (TrueFISP) sequence. This sequence is suited for anatomic studies of intracisternal structures.1 The parameters used in the present study (TR 8.42 ms, TE 4.21 ms, flip angle 80°, voxel size 0.6×0.6×0.6 mm, TA 110 s) depict arterial blood flow with a flow void. The excellent contrast to cerebrospinal fluid allows the measurement of external diameter of vessels in areas where small tortuous vessels become obscured in thick routine slices.

Evaluation of the Imaging Findings
Images were evaluated separately by an experienced neuroradiologist (WF) and neurologist (JK), each blinded to the clinical diagnosis. After the TOF sequence of all patients was evaluated in a maximum intensity projection of the luminal flow, the TrueFISP sequence was independently analyzed in the 3-dimensional data set and on transversal reconstruction. If a vertebrobasilar occlusion was suspected due to the comparison of both sequences, T2-weighted conventional slices and TOF source images were additionally evaluated.

Vessel Grading and Quantification
Diameters of vertebrobasilar arteries were measured in 10-mm distances to the basilar junction. Anomalies of vessel anatomy were graded as follows: Grade 0, normal anatomy; Grade I, hypoplastic or stenosed variants; and Grade II, discontinuity of the vertebrobasilar arteries.

Diagnosis and Classification of Subgroups
Each patient received a thorough neurological examination. Stroke territories were deduced from diffusion-weighted images or, in case...
of older lesions, from T2-weighted images. The vessel territory of transient ischamiases without diffusion-weighted imaged lesions was defined clinically. Patients with a permanent or transient ischemia in the vertebral (VA), basilar, or posterior cerebral artery-supplied areas were classified as “posterior ischemia.” All other patients were classified as “no posterior ischemia.”

Statistical Analysis
Statistical evaluations were carried out computing correlations or performing Student t test or $\chi^2$ tests. The level of significance was set at $P<0.05$. For assessing interobserver agreement, the kappa coefficient was calculated.

Results

Population and Vascular Territories
In total, 104 patients (49 female, 55 male; mean age 65.4 years) were included. Ischemia in the posterior circulation was detected in 30 patients. Seventy-four patients fell in the category “no posterior ischemia,” of which 57% had ischemia in the anterior circulation. The mean ages of the subgroups were not significantly different (65.0 versus 65.7 years).

Figure 1. A patient with a cardiogenic stroke in the posterior circulation and a congenital variant of the vertebrobasilar arteries. A hypoplasia of the right VA (white arrows) is identified by identical grading on TOF angiography (a, c) as well as on TrueFISP imaging (b, d).

Figure 2. A patient with vertebrobasilar TIA and an acquired occlusion of the vertebrobasilar arteries. The right VA is missing only on TOF angiography (a, c), whereas it is clearly depicted (white arrows) by TrueFISP-imaging (b, d).
Quantification of Vessel Diameters
Diameters of the vertebrobasilar arteries were significantly larger in the structural TrueFISP than in the luminal TOF sequence ($P<0.0002$). Using TOF angiography, the average diameter of the arteries was 2.2 mm (VA right), 2.7 mm (VA left), and 3.7 mm (basilar artery). TrueFISP indicated diameters of 2.5 mm, 3.1 mm, and 3.9 mm, respectively.

Detection of Hypo-/Aplastic Variants and Stenoses/Occlusions
Interobserver agreement for grading of vessels was 73% for TOF ($\kappa=0.6$) and 81% for TrueFISP ($\kappa=0.71$). Grading of vessel anomalies differed significantly between TOF and TrueFISP (Grade 0, 52 versus 64 patients; Grade I, 26 versus 34 patients; Grade II, 26 versus 6 patients; $P=0.0013$). The TrueFISP identified hypo- and aplastic vertebrobasilar variants in 6 of 26 patients with TOF angiographically suspected vertebrobasilar occlusion (Figure 1). It confirmed acquired stenosis or occlusion (Figures 2 and 3) with regular exterior diameters in 12 patients in which the TOF sequence demonstrated anomalies of Grade I or II. Additional analysis of conventional images (T2-weighted and TOF source images) depicted the TOF angiographically missing or stenosed vessel segment in only half of these patients. Although anomalies in TrueFISP were equally distributed in the “posterior ischemia” and “no posterior ischemia” subgroups, patients with differing vessel status grading between TOF and TrueFISP were significantly more frequent in the “posterior ischemia” group ($P=0.01$).

Discussion
Recanalization is the primary goal of acute stroke therapy. Therefore, imaging of vessel occlusion is essential for therapeutic implications. However, due to frequent anatomic variants, TOF angiography of vertebrobasilar vessels may be misleading. For an additional effort of less than 2 minutes of scanning time, the TrueFISP sequence potentially allows for distinguishing between acquired alterations and congenital variants of the vertebrobasilar vessels. Hypoplastic vertebral arteries show concordant diameters in both sequences. In contrast, occlusions are represented by a vessel that is visible in the anatomic TrueFISP sequence but invisible in the functional TOF angiography.

The higher frequency of vertebrobasilar occlusions or stenoses in the posterior ischemia group shown by the proposed method confirms earlier results, which correlated vertebrobasilar vessel lesions with posterior stroke incidence. However, in concordance with other studies, inborn variants of the vertebrobasilar anatomy detected by TrueFISP did not predispose to posterior ischemia.

In conclusion, addition of a TrueFISP sequence allows securely differentiating between acute thrombosis and hypoplastic anatomic variants of the vertebrobasilar arteries. Therefore, the method may help to select patients for immediate therapeutic procedures like intra-arterial intervention.

Disclosures
None.

References