Vertebral Artery Hypoplasia Does Not Influence Lesion Size and Clinical Severity in Acute Ischemic Stroke

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Background: Vertebral artery hypoplasia (VAH) is common, but its role in acute ischemic stroke (AIS) is uncertain. We aimed to evaluate the frequency, characteristics, and role of VAH in a large typical cohort of patients with AIS. *Methods:* Up to 815 AIS patients (52.8% men, mean age 70 ± 14 years) were included in the study. All patients received a stroke work-up including brain imaging and duplex ultrasound. VAH was defined by a vessel diameter of less than or equal to 2.5 mm or a difference to the contralateral side of greater than 1:1.7. Vascular risk factors and stroke features were recorded. The subgroup of patients with posterior circulation AIS and magnetic resonance imaging was analyzed additionally, including the parameter of stroke extent. *Results:* Up to 111 patients (13.6%) had VAH, with a mean diameter of 2.4 ± .4 mm. Patients with VAH were significantly younger ($P = .037$) and more often male ($P = .033$). There was no difference considering the National Institutes of Health Stroke Scale and modified Rankin Scale scores on admission or history of stroke. The distribution of patients without VAH was significantly different among the groups with anterior, posterior, and both circulations ischemia ($P = .009$). In the group with posterior circulation stroke, 36 patients (20.9%) had VAH. There were no differences in age, sex, history of stroke, risk factors, vascular territory, stroke size, or etiology. VAH patients had less often embolic stroke patterns ($P = .009$). *Conclusions:* VAH is more common in patients with posterior circulation stroke and in younger patients. Apart from that, we found no clear evidence that VAH would be a predisposing factor for stroke or that it increased the risk for larger ischemic lesions in the posterior circulation. **Key Words:** Vertebral artery hypoplasia—ischemic stroke—posterior circulation—risk factors.

Introduction

Although vertebral artery hypoplasia (VAH) is quite common and known since decades, it still presents a number of uncertainties: There is no generally accepted definition and its role as a stroke risk factor is discussed controversially.[1,2] Data in the literature on this subject are rather rare and inconsistent, as several small studies have used different patient collectives and criteria. While some studies have identified VAH as a risk factor that increases vulnerability for stroke, other authors have come to contrary conclusions.[1-8] Furthermore, most studies have naturally concentrated on the posterior circulation ischemia.

We aimed to evaluate the frequency and role of VAH in a large typical cohort of patients with acute ischemic stroke (AIS) regarding clinical characteristics, risk factors, stroke history, and stroke features. We additionally focused on patients with ischemic stroke in the posterior circulation

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and looked into associations of VAH with stroke pattern, size, and etiology.

Patients and Methods

A total of 815 patients with AIS, consecutively admitted to our Stroke Competence Center within 1 year, were included in this study. The local ethics committee approved the use of the data for the purpose of this study. The diagnosis of AIS was made by a neurologist after history taking, neurological examination, and cerebral imaging (magnetic resonance imaging [MRI] or computed tomography), according to the standard procedure. All patients were admitted to our Stroke Unit and underwent thorough neurological examination, including National Institutes of Health Stroke Scale (NIHSS) and modified Rankin Scale (mRS) scoring. Duplex ultrasound of the vertebral arteries (segments V1 and V2) was performed with a Philips iU22 ultrasound system with a 9-3 MHz linear array transducer (Philips Ultrasound, Bothell, WA, USA).

Diameters of the vertebral arteries were determined in the V2 segment of the vertebral artery, as reported in the literature.[2] VAH was defined by a vessel diameter of less than or equal to 2.5 mm or a difference to the contralateral side of greater than 1:1.7, to be in line with previous relevant work.[2] VAH in patients with posterior circulation stroke was confirmed by MRI, in order not to misdiagnose a possible vertebral artery dissection. These patients were additionally evaluated in a separate analysis.

MRI was performed on 1.5 or 3 Tesla Siemens MR scanners (Siemens Medical Systems, Erlangen, Germany) with echo planar hardware using a standardized stroke protocol including transverse, coronal, and sagittal localizing sequences; proton density and T2-weighted images (including fluid-attenuated inversion recovery); T1-weighted images; susceptibility-weighted gradient-echo images; and diffusion-weighted images and three-dimensional time-of-flight magnetic resonance angiography (MRA) sequence of the circle of Willis and the neck arteries in coronal direction. In cases of suspected vertebral artery dissection, axial fat suppressed T1-weighted images were performed, as suggested in the literature.[9]

Cerebro- and cardiovascular risk factors (arterial hypertension, diabetes, hyperlipidemia, smoking, atrial fibrillation, and history of coronary heart disease) as well as history of cerebrovascular disease (ischemic or hemorrhagic stroke, transient ischemic attack) were recorded.

The pattern (embolic, territorial, lacunar) and the vascular territory (anterior, posterior, or both circulations) of the ischemic lesions were also recorded. In the group of patients with AIS in the posterior circulation, stroke territory was classified in vertebral or basilar artery territory, posterior cerebral artery territory, or both (in case of multiple infarcts). The size of ischemic lesion (<1/3, 1/3, 2/3, or >2/3 of the vascular territory) and its etiology (macroangiopathic, microangiopathic, cardioembolic, other, or undetermined) were included in the analysis.

Distribution of frequencies was analyzed using the χ^2 test or Fisher's exact test where appropriate. Group comparisons for metric data were analyzed using the Student's *t*-test and for ordinal data using the Mann–Whitney *U*-test. We applied a Bonferroni correction for multiple comparisons in order to compensate for a type 1 error. Statistical significance was assumed when $P < .05$. Statistical analysis was performed using SPSS, version 22 (IBM, Armonk, NY, USA).

Results

About half of the 815 patients included in the study were men (430; 52.8%). Mean age was 70 ± 14 years. The general characteristics of all patients are presented in Table 1. Median NIHSS score on admission was 4 (range 0-28) and median mRS score was 4 (range 0-5). Ischemic lesions were mostly localized in the anterior circulation (566; 69.4%), followed by the posterior circulation (205; 25.2%) and both circulations simultaneously (43; 5.3%). VAH was present in 111 patients (13.6%).

Table 2 summarizes the characteristics of the VAH. The mean vertebral artery diameter in VAH was 2.4 ± .4 mm, as opposed to the mean diameter of the nonhypoplastic vertebral arteries (3.7 ± 2.4 mm) and the overall mean diameter of all vertebral arteries in the study (3.1 ± .9 mm). The mean diameter ratio was .66 ± .16. VAH was located on the right side in 67 (60.4%) patients, on the left side

Table 1. *General characteristics of all patients*

Number of patients	815
Mean age (years ± SD)	70 ± 14
Men	430 (52.8%)
Women	385 (47.2%)
Median NIHSS score on admission (range)	4 (0-28)
Median mRS score on admission (range)	4 (0-5)
Vertebral artery hypoplasia	111 (13.6%)
Risk factors	
Arterial hypertension	657 (80.6%)
Diabetes	228 (28%)
Hyperlipidemia	378 (46.4%)
Smoking	143 (17.5%)
Coronary heart disease	103 (12.6%)
Atrial fibrillation	220 (27%)
History of cerebrovascular disease	
History of ischemic stroke	136 (16.7%)
History of hemorrhagic stroke	14 (1.7%)
History of TIA	18 (2.2%)
Stroke localization	
Anterior circulation	566 (69.4%)
Posterior circulation	205 (25.2%)
Anterior and posterior circulations	43 (5.3%)

Abbreviations: mRS, modified Rankin Scale; NIHSS, National Institutes of Health Stroke Scale; SD, standard deviation; TIA, transient ischemic attack.