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Vertebral artery hypoplasia and the posterior circulation stroke

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Summary The aim of this preliminary study is to evaluate the hypothesis of a possible causal link between the anatomical findings of vertebral artery hypoplasia (VAH) and the incidence of posterior circulation stroke. We used full ultrasonographic examination to evaluate patients with stroke in the vertebrobasilar circulation territory over a period of 1.5 years. The diameter equal or less than 2.5 mm (in V1 and V2 segment of the vertebral artery) was set as a feature of vertebral artery hypoplasia. Magnetic resonance imaging and angiography (MRI and MRA) or computed tomography and angiography (CT and CTA) were performed to confirm the anatomic variation of hypoplasia and the site of the cerebral ischemic territory. In the group of 44 stroke patients, 9 (20%) had a hypoplastic vertebral artery and 35 (80%) were without VAH. Although vertebral artery hypoplasia in previously published literature is seldom shown as a leading risk factor for stroke in vertebrobasilar (posterior) circulation, its occurrence is not negligible and in coexistence with known risk factors of stroke may increase the negative clinical impact. Vertebral artery hypoplasia can be diagnosed non-invasively with duplex ultrasonography. It is therefore a useful method for detection of this anatomic variation and for follow-up examination.

Introduction

The vertebral artery (VA) as a part of the vertebrobasilar cerebral circulation is one of the main branches of the subclavian artery. The course of the VA is divided into 4 sections [1,2]. It originates as section V0 from the posteromedial part of the arc of the subclavian artery and continues cranially. It is followed by the prevertebral segment (V1), which in 90% enters into the costotransverse foramen of the sixth cervical vertebra (C6). Variations as entrance in the C5 or above the C6 vertebra, coiling or kinking of the vertebral artery can occur. The intervertebral segment (V2) passes through the costotransverse canal of the cervical vertebrae up to the C2 vertebra. The atlas loop segment (V3) is created by a curved course of the artery around the atlas. The intracranial segment V4 is the section of the vertebral artery after penetrating the atlantooccipital membrane, dura mater and arachnoidea. At the clivus the right and left vertebral artery merge to form the basilar artery, which is a part of the intracranial posterior circulation.

The diameter of vertebral arteries varies from 1.5 to 5.0 mm. Identical width of VA occurs in 25% of the population, in 65% the left vertebral artery is wider, whereas in the remaining 10% the right vertebral artery is larger [3]. Khan et al. found dominance of the left vertebral artery in 50%, and of the right vertebral artery in 25% in regard to the diameter of the vessel [4].

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The following congenital anatomic variations of the vertebral artery are described in the literature: vertebral artery aplasia and vertebral artery hypoplasia (VAH). Aplasia of VA occurs in about 1% of the population [5].

Vertebral artery hypoplasia (VAH) is classified as a vessel with a diameter in the entire course of less than 2 mm [6], respectively less than 3 mm [7], or with a side difference equal or greater than 1:1.7 [8]. Additionally to the vessel diameter, another criterion contains reduced blood flow velocity and increased resistance index values in the ultrasonographic findings [1,9]. There is a tendency of compensatory increase in the vessel diameter of the contralateral vertebral artery of more than 5 mm [1]. These various definitions of the incidence of VAH are based on subsequent characteristics: a diameter of less than 2 mm was observed by the method of duplex ultrasonography by the authors Delcker and Diener in 1.9% of the population [6], a diameter of less than 3 mm was described by Touboul et al. in 6% of the population [7]. Trattnig et al. set a side asymmetry in the ratio 1:1.7 for more than 10% of patients examined by ultrasonography [8]. Frequency of VAH (diameter equal or less than 2 mm) in the general population is 26.5% in unilateral and 1.6% in bilateral hypoplasia of the vertebral artery [10]. In terms of side difference, the right hypoplastic vertebral artery occurs in 6.2% of the population, while left vertebral hypoplasia is present less frequently in 4.5% [2]. Visualization of vertebral artery is possible by ultrasonographic examination, by invasive or non-invasive angiography (MRA, CTA), and also by autopsy findings. As mentioned previously, a more narrow vessel lumen is present in the ultrasonographic image in vertebral artery hypoplasia, and additionally, blood flow parameters are defined by a reduced diastolic flow velocity associated with higher peripheral resistance. The resistance index (RI) is equal to or greater than 0.75. The peak systolic velocity (PSV) is usually less than 40 cm/s [1,5].

In the literature, morphological variations of the vertebral artery are described as being associated with different clinical symptoms. Nevertheless, vertebral hypoplasia as a possible risk factor for pathology, particularly of stroke in the vertebrobasilar circulation territory, was little emphasized yet.

The aim of this preliminary study is to evaluate a hypothesis of a possible causal link between the anatomical findings of VAH and the incidence of posterior circulation stroke. For this purpose, we assessed the relative frequencies of posterior circulation strokes in patients with VAH as compared to patients without VAH, and also the relative frequencies of the conventional vascular risk factors (hypertension, diabetes, hyperlipidemia and smoking). Additionally, we determined the possible mechanism of stroke in our patients.

Materials and methods

A group of 44 patients (30 men, 14 women; mean age 67 years [range 44—88]) with acute ischemia in the vertebrobasilar territory had a full ultrasonographic examination of the extra- and intracranial arteries between September 2009 and February 2011.

The location of the acute ischemic infarct was judged clinically and confirmed by CT scan or MRI.

We excluded patients with transient ischemic attacks (TIA), patients with other vertebral artery findings (such as atheromatosis, stenosis or occlusion) or other cerebral lesions, as well as those in whom a full ultrasonographic examination of the vertebral arteries was not possible.

We used a 7.5-MHz linear array transducer for the duplex ultrasonographic examination of the vertebral arteries (B-mode and color-coded duplex flow imaging). In the V1 (prevertebral) and V2 (intervertebral) segments of the extracranial vertebral artery the distance between the internal layers of the parallel walls of the vessel (caliber of VA) and the hemodynamic characteristics of blood flow were measured.

The diameter equal or less than 2.5 mm, respectively the side difference equal or greater than 1:1.7 were set as a feature of vertebral artery hypoplasia. Additionally, reduced flow velocities as compared to the contralateral side, and higher peripheral resistance ipsilaterally (RI equal or greater than 0.75) were considered.

MRA, CTA or conventional angiography was performed to confirm the presence or absence of the anatomic variation of hypoplasia.

We also investigated the occurrence of other concomitant vascular risk factors such as hypertension, diabetes, hyperlipidemia and smoking.

Results

In the group of 44 posterior circulation stroke patients, 9 (20%) had a hypoplastic vertebral artery and 35 (80%) were without VAH (Fig. 1A). There was more frequent right-sided VAH in 7 (78%), as compared to left-sided VAH in 2 (22%) cases (Fig. 1B). One patient had bilateral VAH (both vertebral arteries had a diameter of less than 2.5 mm), more significant on the right side. None of the patients had basilar artery hypoplasia. In the group of non-VAH patients were 22 men and 13 women, in the VAH group 8 men and 1 woman (Fig. 1C). There was a slight difference for age between the non-VAH (mean age 68.3 years) and VAH group (mean age 62.3 years).

The distribution of other vascular risk factors in both groups was represented as follows (Fig. 1D): hypertension ($n = 40$ patients), diabetes mellitus ($n = 19$), hyperlipidemia ($n = 17$) and smoking ($n = 16$).

The frequency of the presence of these risk factors (hypertension: $p = 0.99$; diabetes mellitus: $p = 0.26$ and smoking: $p = 0.45$) in patients with posterior circulation strokes with or without VAH did not differ.

We found that in the group of patients without VAH hyperlipidemia occurred more often than in the VAH group (16:1). There was a statistically significant relationship between finding of non-VAH and hyperlipidemia ($p = 0.027$).

Possible mechanism of stroke were embolism, especially cardioembolism ($n = 10$), atherosclerotic changes of vessels (small vessel disease $n = 16$, or large vessel disease $n = 25$). In 6 cases, the mechanism of stroke was cryptogenic (unknown mechanism $n = 6$) (Fig. 1E).