The Frequency and Significance of the Vertebral Artery Hypoplasia in Posterior Circulation Stroke

Ž Biljana[1], S Jelena[1], S Petar[2,3], S Dragan[4,5], P Sladjana[4,5], A-T Aleksandra[4]

ABSTRACT

Objective: Vertebral artery hypoplasia (VAH) is an uncommon embryonic variation of posterior (vertebrobasilar) circulation. The literature reports on its incidence in general population are in the range between 2 and 10%. The aim of the paper was to analyze the frequency and association between VAH and posterior circulation stroke (PCS). Besides VAH, other risk factors responsible for PCS were examined.

Methods: The study was a prospective one, conducted at the Neurology Clinic, Clinical Center Niš. It enrolled 50 patients with the established diagnosis of PCS. The control group consisted of 40 patients without PCS. The presence of VAH was determined by performing Color-Doppler Echosonography (CDE) and proved by computed tomography angiography (CTA).

Results: Our results showed statistically significant presence of VAH in patients with PCS in comparison to the controls (38%: 10%).VAH was found to be an independent risk factor for PCS onset. The presence of VAH in the group of patients with posterior circulation stroke in relation to other patients from the experimental group without VAH is a poor prognostic parameter concerning the severity of clinical manifestation and the outcome of the disease.

Conclusion: VAH is a risk factor for posterior circulation stroke onset. Its presence in patients with posterior circulation stroke is a poor prognostic parameter significantly correlating with the severity of clinical presentation and higher mortality rate.

Keywords: Circulation stroke, posterior circulation stroke, vertebral artery hypoplasia

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From: [1]Clinic of Neurology, Clinical Center Nis, Serbia, [2]Clinic of Neurology, Clinical Center Vojvodine, Novi Sad, Serbia, [3]Faculty of Medicine, University of Novi Sad, Serbia, [4]Center for Radiology, Clinical Center Nis, Serbia. [5]Faculty of Medicine, University of Nis, Serbia.

Correspondence: Dr Ž Biljana, Clinic of Neurology, Clinical Center Nis, Serbia.
E-mail: zivadinovicbilja@gmail.com

INTRODUCTION

The vertebral artery, arteria vertebralis (VA), is formed in the embryo of the longitudinal vascular anastomoses between parallel intersegmental arteries and seven cervical intersegmental arteries that arise from paired dorsal aortas and supply the spinal cord and eight pairs of cervical nerves. Seven proatlantal intersegmental arteries involute and usually become the proximal part of the VA and subclavian artery (SA). Anomalies that may occur are numerous:

The type I proatlantal-intersegmental artery arises from the internal carotid artery (ICA) andsuboccipitally binds to VA, ascends and curves, then penetrates the skull between the occiput and atlas and further follows the normal course of VA.

The type II proatlantal artery arises from the ECA and is usually associated with VA agenesis and hypoplasia of some VA segments.

A persistent hypoglossal artery that enters the skull through the anterior condyloid foramen is often accompanied by complete occlusion of proximal VA. First cervical intersegmental artery may persist as suboccipital intersegmental artery (1, 2). There are many other variations of fetal circulation with or without communication between vertebrobasilar and carotid territory, thus affecting redistribution of blood in cerebral circulation (3-7).

Vertebral artery hypoplasia (VAH) was first described in the 19th century. It is an uncommon embryonic variation of posterior circulation. There is no absolute consensus on VAH definition. The actual definition classifies VAH as a vessel with a diameter of 2mm and less (rarely 3mm≤), with side difference of 1:1.7 ≥ (7). Its incidence has been described in 2-6% of cases in general population. Some authors report the percentage between 10 and 15% (8, 9, 10).

As for localization, right-sided VAH is more common, but literature data reveal different percentage concerning the localization. The results of one group of authors show that the frequency of right-sided VAH is twice as more common than left-sided VAH. On the other hand, some authors also describe the dominance of the right VAH in comparison to left-sided VAH, but the difference in percentage is less [6.2%: 4.6%] (9, 10).

Besides vessel diameter measurements it is important to measure hemodynamic parameters as well. Some papers report systolic velocity decrease below 40cm/sec and resistant index increase (RI) >0.75 n patients with VAH (10).

Aim of this paper was to examine the incidence and significance of VAH presence in patients with PCS and determine the relationship between VAH and the clinical finding, clinical manifestation and final outcome of the patients with proven PCS.

METHODS

The study was conducted as a prospective one at the Neurology Clinic. It enrolled 50 patients hospitalized at the Clinic with diagnosed PCS. The control group consisted of 40 patients without PCS diagnosis.

The diagnosis of PCS was determined by CT or MRI findings. All the patients underwent AV ultrasound, observing artery V1 and V2 segments with a linear probe 7.5MHz at B-mode and Color Doppler sonography.

The values of vessel diameter ≤ 2mm were considered as hypoplastic vessel findings. We followed flow velocity and resistance index (IR). VAH was confirmed by computed tomography angiography (CTA) method.

RESULTS

The mean age of patients in experimental group (67.26±11.01) was higher than in the control group (39.15±9.19), with statistically significant difference (t=13.201; p<0,0). As for gender distribution, there were more male patients (66%) in the experimental group and more female patients (62.5%) in the control group.

Vertebral artery hypoplasia was registered in 38% of patients with posterior cerebral stroke in the experimental group and in 10% of patients in the control group, what is statistically significant difference at the level p<0.01 (χ^2 =9.158; p=0.002).

Right side VAH is dominant both in experimental and control group

Increasing age, male gender, VAH, HTA, hyperlipidemia and diabetes were predictors independently associated with the stroke. The presence of VAH was found to be an independent risk factor for posterior circulation stroke onset (OR=5.516; 9%CI=1.694-17.957; p=0.005).

Based on the odds ratio it can be seen that age as a continuous variable increases the possibility of posterior circulation infarction for 30% (OR=1.281; 9%CI=1.154-1.422; p<0.001). Female gender is statistically significant protective factor (OR=0.309; 9%CI=0.130-0.736; p=0.008).

Hypertension was also identified as a very strong risk factor (OR=15.691; 9%CI=5.574-44.174; p<0.001), as well as hyperlipidemia (OR=6.152; 95%CI=2.450-15.446; p<0.001) and diabetes (OR=6.937; 95%CI=1.871-25.728; p=0.004).Other analyzed factors were not of statistical significance for posterior circulation infarction onset.

The number of patients with more severe clinical manifestations is significantly higher in the group of patients with VAH.

The percentage of lethal outcomes in the experimental group in patients with VAH is statistically significantly higher in comparison to patients without VAH (χ^2 =3.34, p= 0.05).

DISCUSSION

Because of its specific anatomic location and inconvenient access for surgical procedures, VA was long inadequately perceived in conservative and surgical treatment (10, 11).

Since EgazMonis performed the first vertebral angiography in 1993, this blood vessel has become more and more important, as well as its significant involvement in the pathology of the head, neck and cerebral circulation. The very aim of our prospective study was to examine the correlation between VAH and posterior circulation infarction.

As for age distribution, the patients in the experimental group were older in comparison to the control group. However, since the aim of the study was to determine the incidence of VAH which is an inborn anomaly of the blood vessel and its frequency is unaffected by the age of patients, such age distribution does not affect group homogeneity.

Concerning the localization, our study results showed that VAH was most commonly found on the right side, 52.6% in the experimental group and 75% in the control group (table 2). Such a distribution of VAH is logical, since the left vertebral artery is dominant in world's population (50-75%). Right vertebral artery is dominant in 25% and the two vertebral arteries are of the same caliber in about 25% (9, 10).

The criterion for hypoplasia of the vertebral artery in our study was the diameter of the artery ≤2mm and vertebral artery flow volume less than 30-40ml/min (9, 10).

Some authors considered a vertebral artery diameter of 2.2mm or ≤3mm to indicate vertebral artery hypoplasia. Although greater incidence of VAH would be expected with greater diameter of the blood vessel, the percentage of VAH incidence reported by these authors is the same, accounting for about 6%. (9).

The data from the studies that examine the relationship between VAH and posterior cerebral infarction show the presence of VAH in different numbers: 20%- 72.72% of patients with infarction at this localization (10-14). Our investigation of patients in the experimental group also registered VAH more commonly than in the control group, but the percentage is slightly lower, 38%: 10 % .Table 2.

Table 2 shows that VAH, along with age, male gender, hypertension, hyperlipidemia and diabetes, was found to be an independent risk factor for posterior cerebral infarction (13). Its presence is associated with five-fold increased risk of PCS.

As for the severity of clinical manifestation and final outcome of the disease, VAH is an unfavourable prognostic parameter in patients with posterior cerebral stroke. The Tables 4 and 5 show that in these patients NIHSS score is expected to be higher and the final outcome of the disease poor.

CONCLUSION

The presence of VAH is a risk factor for posterior cerebral infarction onset and a poor prognostic parameter in patients with posterior cerebral infarction, correlating with the severity of clinical manifestation and higher percentage of lethal outcomes.

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Table 1- Distribution of subjects in experimental and control group in relation to the presence of VAH

VAH	Experimental group	Percentage	Control group	Percentage	Total
No	31	62 %	36	90 %	67
Yes	19	38 %	4	10 %	23
Total	50	100 %	40	100 %	90

Table 2: Distribution of subjects in both experimental and control group with VAH compared to VAH localization

VAH localization	Experimental group	Percentage	Control group	Percentage	Total
Left	6	31.16 %	1	25 %	7
Right	10	52.6 %	3	75 %	13
Both	3	15.8%	0	0	3
Total	19	100 %	4	100 %	23

Table 3: VAH as a risk factor for posterior cerebral stroke onset in comparison to other risk factors (univariate logistic model)

	OR	P
Age	1.281	<0,001
Female gender	0.309	0.008
VAH	5.516	0.005
HTA	15.691	<0.001
Hyperlipidemia	6.152	<0.001
DM	6.937	0.004
Arit.aps	1.176	0.852
IM	5.318	0.130
Cardiomyopathy	1.131	0.767
Hemat.disease.	2.489	0.438
Malignancies	2.498	0.411
Other risk factors	1.700	0.857

Table 4: Distribution of VAH + and VAH- patients from the experimental group in relation to the values of NIHSS score

Score	Number of VAH+	Percentage of VAH+	Number of VAH-	Percentage of VAH -	Total number	Total percentage
< 4	10	52.63 %	22	70.96%	32	64 %
> 4	9	47.36 %	9	29.03%	18	36 %
Total	19	100%	31	100%	50	100%

Table 5: Distribution of VAH + and VAH – patients from the experimental group in relation to the outcome of the disease

Outcome	Number of VAH+	Percentage of VAH+	Number of VAH -	Percentage of VAH -	Total number	Total percentage
Survived	14	73.68 %	30	96.77 %	44	88 %
Deceased	5	26.32 %	1	3.22 %	6	12 %
Total	19	100 %	31	100 %	50	100 %