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Original Research Article

The prevalence, clinical characteristics, and brain MRI changes in intracranial artery hypoplasia: a retrospective single-center cross-sectional study

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ABSTRACT

Background: Cerebral artery hypoplasia (CAH) has recently been recognized as potential risk factor of stroke due to reduced cerebral blood flow, increased atherosclerosis, and aneurysm formation. This study aimed to describe the profile of vertebral artery hypoplasia (VAH), anterior CAH (ACAH), and posterior CAH (PCAH) in symptomatic subjects.
Methods: This was a retrospective cross-sectional study using medical record in a secondary private hospital in Jakarta, Indonesia, in January-December 2022. The inclusion criteria were age ≥18 years with neurological symptoms, underwent brain MRI, and being diagnosed with CAH using DSA. The exclusion criteria were findings of other vascular pathologies including dissection and >50% intracranial vessel stenosis. Demographics of age, sex, body mass index (BMI), hypertension, diabetes, cardiac disorder, and previous stroke were analyzed among VAH, PCAH, and ACAH groups.
Results: Of 769 subjects with clinical symptoms undergoing DSA, there were 66 (8.6%) cases of intracranial artery hypoplasia, including VAH (4.6%), PCAH (1.2%), and ACAH (2.9%). Subjects were predominantly old (53.2±10.1 years), male (53.0%), with BMI of 24.9±3.9 kg/m^2 and hypertension (69.7%). Previous stroke (69.7%) was more prevalent than in previous study (28.1%). Stroke and brain ischemic lesion were detected in 89.4% and 84.8% cases. No differences were found in all parameters among all groups, but posterior circulation symptoms tended to be found in VAH (28.1%) than general stroke (20.3%).
Conclusions: The high percentage of recurrent stroke and corresponding clinical symptoms associated with CAH supported that CAH may be a risk factor for clinical symptoms, including stroke, regardless of the involved artery.

Keywords: Anterior cerebral artery hypoplasia, Cerebral artery hypoplasia, Posterior cerebral artery hypoplasia, Stroke, Vertebral artery hypoplasia

INTRODUCTION

Ischemic stroke is still one of the most debilitating diseases in the world, including Indonesia. The National Basic Health Research of Indonesia in 2018 has reported the prevalence of stroke to be 10.9 per 1000 population, of which 8.6% occurred at subjects of less than 45 years old. Stroke may contribute to reduced productivity, increased dependency, and increased healthcare costs.[1]

Our brain has sophisticated vascularization which stems from anterior circulation system of a pair of carotid arteries and posterior circulation system of a pair of vertebral arteries. Disturbance in cerebral blood flow may be compensated by the collaterals, including the circle of Willis that connects the two circulation systems.[2]

Congenital variation of vertebral arteries is common, including vertebral artery hypoplasia. Vertebral artery

hypoplasia (VAH) was reported to be 1.9-≤26.5% in prevalence and can be recognized in healthy individuals. While VAH was firstly considered as a non-dangerous anatomic variant, recent studies have associated its presence with increased predisposition for posterior circulation stroke, especially involving the posterior inferior cerebellar artery and lateral medullary infarcts, and when accompanied with other atherosclerotic risk factors.[2-4] Unilateral VAH may be associated with ipsilateral atherosclerosis of VA, relative hypoperfusion in ipsilateral PICA, and increased risk of contralateral cardioemboli whereas bilateral VAH may present with the involvement of multiple bilateral vascular teritories.[4]

The most common types of anatomical variation in the circle of Willis includes hypoplasia of one or both of posterior communicating arteries (34-68%), hypoplasia of A1 segment of anterior cerebral artery (4-10%), absence of anterior communicating artery (12-21%), persistent fetal origin of posterior cerebral artery (4-26%), and infundibular dilatation of posterior communicating artery (7-15%). Anterior cerebral artery hypoplasia variant of the circle of Willis is uncommon, with a frequency of 1-13% in another study. Ischemic strokes involving the ACA territory were also uncommon (0.6-3%).[5] Despite rarer in publications than VAH, ACAH of A1 segment of the circle of Willis has also been reported as a potential risk factor for stroke, especially in those without intact collateral circulation.[5,6] In addition, some reports described that ACAH may increase the collateral blood flow in anterior communicating artery and predispose to aneurysm formation.[7] Another study described that anatomical variation in the circle of Willis was associated with 1.38 times to develop ischemic stroke.[7]

This study aimed to describe the demographics, clinical, and brain MRI profile of VAH, ACAH, and posterior cerebral artery hypoplasia (PCAH). To our knowledge, this is the first study assessing hypoplasia using digital subtraction angiography (DSA), which is the gold standard for assessing vascular anomalies. Hypoplasia was also being studied in its relation to overt clinical symptoms and subtle changes of brain MRI ischemic lesion. The evidence in this study is expected to add value in understanding hypoplasia and its potential risk.

METHODS

This was a retrospective single-center study conducted in a secondary private hospital in Jakarta, Indonesia, in January to December 2022.

Inclusion and exclusion criteria

Adults of ≥18 years old with neurological symptoms including headache, vertigo, or associated with stroke, underwent brain MRI, and being diagnosed with cerebral artery hypoplasia using DSA were included in this study. The exclusion criteria included the findings of other vascular pathologies including vertebral artery dissection and >50% stenosis of intracranial vessel, as well as the unavailability of DSA and MRI expertise data.

Total sampling within the study duration was performed in this study due to relatively small percentage of prevalence reported in previous studies.

Vertebral artery hypoplasia was defined with diameter of VA of ≤2 mm at V4 segment whereas anterior cerebral artery hypoplasia (ACAH) and posterior cerebral artery hypoplasia (PCAH) were defined with diameter of <1mm at the circle of Willis.[2,8] The data were documented from the DSA expertise report.

The information recorded in this study were demographics including age, gender, and body mass index (BMI), comorbidities of hypertension, diabetes, dyslipidemia, and cardiac disorders, clinical symptoms, and brain MRI abnormalities of ischemic lesion. Body mass index was further categorized based on the Asian categorization into underweight (BMI<18.5 kg/m^2), normoweight (BMI 18.5-22.9 kg/m^2), overweight (BMI 23.0-24.9 kg/m^2), obese grade 1 (BMI 25.0-29.9 kg/m^2), and obese grade 2 (BMI≥30 kg/m^2). Clinical symptoms were categorized into nonstroke (headache, epilepsy, etc.) and stroke, which was further analyzed into posterior circulation symptoms (vertigo) or not. Stroke was defined as clinical syndrome of sudden focal or global neurological deficits due to brain, retina, or spinal cord ischemia that can only be explained by vascular etiology. Posterior circulation symptoms included isolated homonymous hemianopia, brainstem syndrome, or cerebellar symptoms including vertigo. The clinical symptoms in this study were documented retrospectively from the medical record. Brain MRI result of ischemic lesion was also documented retrospectively from the expertise report.

Data were presented in percentage for categorical variables, in mean for normally distributed numeric variables, or in median for abnormally distributed numeric variables. Bivariate analysis of clinicoradiological profile and hypoplasia was carried out using chi square, Fisher, independent t-test, or Mann-Whitney test as appropriate. Statistical significance was performed two-sided and determined with $p<0.05$.

RESULTS

Of 769 subjects who underwent DSA due to any clinical symptoms, the prevalence of cerebral artery hypoplasia was 66 (8.6%) subjects. Cerebral artery hypoplasia in this study was mostly VAH (4.6%), followed with ACAH (2.9%) and PCAH (1.2%). The subjects of cerebral artery hypoplasia in this study were predominantly older adult, overweight, male, with comorbidities of hypertension (69.7%) and previous stroke (69.7%). There were only 18.2% subjects with diabetes and no subject with cardiac disorder. Most subjects had recent stroke (89.4%), a-fourth of which presented with vertigo or posterior

circulation symptoms. The symptoms of stroke in this study were 44 (74.6%) subjects hemiparesis and cranial nerve palsy and 15 (25.4%) subjects with vertigo. The nonstroke symptoms included six (85.7%) subjects with headache and one subject (14.3%) with epilepsy. Ischemic lesions were detected in 84.8% subjects (Table 1).

Table 1: Demographics of this study.

Variables	Total (n=66)	Stroke (n=59)	Nonstroke (n=7)
Age (years)	53.2±10.1		
Male n (%)	35 (53.0)	31 (52.5)	4 (57.1)
Body mass index (kg/m^2)	24.9±3.9		
Underweight n (%)	2 (3.0)	2 (3.4)	0 (0.0)
Normoweight n (%)	15 (22.7)	12 (20.3)	3 (42.9)
Overweight n (%)	22 (33.3)	19 (32.2)	3 (42.9)
Obesity n (%)	27 (30.9)	26 (44.1)	7 (11.1)
Risk factors n (%)			
Previous stroke	46 (69.7)	45 (76.3)	1 (14.3)
Hypertension	46 (69.7)	44 (74.6)	2 (28.6)
Diabetes	12 (18.2)	12 (20.3)	0 (0.0)
Clinical symptoms n (%)			
Nonstroke	7 (10.6)		
Stroke	59 (89.4)		
Posterior circulation symptoms	15 (25.4)		
Anterior circulation symptoms	44 (74.6)		
Ancillary findings n (%)			
MRI: ischemic lesion	56 (84.8)	53 (89.8)	3 (42.9)
DSA			
ACA hypoplasia	22 (33.3)	21 (35.6)	1 (14.3)
PCA hypoplasia	9 (13.6)	6 (10.2)	3 (42.9)
VA hypoplasia	35 (53.0)	32 (54.2)	3 (42.9)

Table 2: Demographics of subgroups of hypoplasia.

Demographics	VAH (n=35)	PCAH (n=9)	ACAH (n=22)	VAH versus PCAH	VAH versus ACAH	PCAH versus ACAH
Age (years)	54.0±11.0	50.2±12.7	53.1±7.5	0.38	0.73	0.44
Male	18 (51.4%)	7 (77.8%)	10 (45.5%)	0.26	0.66	0.13
Body mass index (kg/m^2)	25.4±3.4	24.8±4.2	25.6±3.0	0.90	0.39	0.54
Underweight	2 (5.7%)	0 (0.0%)	0 (0.0%)	-	-	-
Normoweight	9 (25.7%)	3 (33.3%)	3 (13.6%)	Ref	Ref	Ref
Overweight	9 (25.7%)	4 (44.4%)	9 (40.9%)	1.00	0.26	0.62
Obesity	15 (42.8%)	2 (22.2%)	10 (45.5%)			
Risk factors						
Previous stroke	27 (77.1%)	4 (44.4%)	15 (68.2%)	0.10	0.45	0.25
Hypertension	26 (74.3%)	5 (55.6%)	15 (68.2%)	0.41	0.62	0.68
Diabetes	8 (22.9%)	1 (11.1%)	3 (13.6%)	0.66	0.50	1.00

Based on the types of cerebral artery hypoplasia, there were no significant differences in the profile of demographics and risk factors in VAH, PCAH, and ACAH. The subjects in all groups had similar age of older adult with similar BMI. The subjects in VAH and ACAH tended to have a higher proportion of history of stroke and hypertension than those in PCAH. The subjects in VAH also tended to have higher proportion of diabetes than those in other groups. On the other hand, male was more commonly found in PCAH (Table 2).

From the clinical findings, there were also no significant differences among groups. The subjects in VAH and ACAH group tended to have a higher proportion of stroke than those in PCAH. In addition, subjects in VAH and PCAH group had higher percentage posterior circulation symptoms than those in ACAH, albeit

insignificant (Table 4). Brain MRI ischemic lesion was also more commonly found in VAH and ACAH than in PCAH subjects (Table 3). Multivariate analysis was carried out using binary logistic regression and no significant interactions were found.

Table 3: Ancillary findings of subgroups of hypoplasia.

Clinical findings	Brain MRI changes								
	VAH (n=35)		P	PCAH (n=9)		p	ACAH (n=22)		P
	Ischemic	Non-ischemic		Ischemic	Non-ischemic		Ischemic	Non-ischemic	
Nonstroke	2 (66.7%)	1 (33.3%)	0.136	1 (33.3%)	2 (66.7%)	0.524	0 (0.0%)	1 (100.0%)	0.136
Stroke	30 (93.8%)	2 (6.2%)		4 (66.7%)	2 (33.3%)		19 (90.5%)	2 (9.5%)	

Table 4: Clinical findings of subgroups of hypoplasia.

Clinical Findings	VAH (n=35)	PCAH (n=9)	ACAH (n=22)	VAH versus PCAH	VAH versus ACAH	PCAH versus ACAH
Nonstroke	3 (8.6%)	3 (33.3%)	1 (4.5%)	Ref	Ref	Ref
Stroke	32 (91.4%)	6 (66.7%)	21 (95.5%)	0.09	1.00	0.06
Posterior circulation	9 (28.1%)	3 (50.0%)	3 (14.3%)	0.36	0.32	0.10
Anterior circulation	23 (71.9%)	3 (50.0%)	18 (85.7%)	Ref	Ref	Ref

DISCUSSION

To our knowledge, this was the first study in Indonesia that describe the prevalence of cerebral artery hypoplasia in subjects with any symptomatic clinical manifestation using the gold standard of DSA. This study discovered that (1) the prevalence of cerebral artery hypoplasia was 8.6%; (2) VAH is the most common type of cerebral artery hypoplasia; (3) despite other similar clinicodemographic profile between this study and general stroke profile, cerebral artery hypoplasia had a higher prevalence of previous stroke history (69.7%) than general stroke population (28.1%); (4) no significant difference among all clinicodemographic characteristics in all types of cerebral artery hypoplasia. This study raised concerns for not only VAH but also any types of cerebral artery hypoplasia as a potential risk factor for symptomatic clinical manifestation including stroke.

The prevalence of all types of cerebral artery hypoplasia in this study was 8.6%, most of which were VAH (4.6%). This number was lower than those reported in previous studies, which ranges from 10.8-43.5%.[8] The difference may be explained by the different ethnicity and the study population. The single-center secondary private hospital setting in this study may not represent the more severe case that has already been referred to the tertiary hospital. In addition, not every subject had been offered DSA in clinical practice. However, this study utilized DSA as the best modality to detect cerebral vessel abnormalities. This study also supported the finding from previous reports that VAH was the most common hypoplasia.

Subjects in this study were mostly male (53.0%) with the age of 53.2±10.1 years, hypertension (69.7%), previous stroke (69.7%), and overweight-to-obese type I (BMI 24.9±3.9 kg/m^2). Similar findings of demographics were found in each subgroup of VAH, PCAH, and ACAH. A similar study from Erdal et al in Turkey recruited 74 cases of acute posterior circulation stroke with VAH, most of which were female (68.9%) with the mean age of 65.4±10.6 years and comorbidities of hypertension (64.9%), hyperlipidemia (64.9%), diabetes (35.1%), and coronary heart disease (21.6%). There were also no differences when the demographics were compared with non-VAH cases.[3] The general demographic profile of stroke in Indonesia had been reported by Stevano et al (2023), which was male (60.8%) of 60.55±13.54 years old with hypertension (77.8%), diabetes (36.6%), dyslipidemia (31.4%), previous stroke history (28.1%), obesity (19.6%), coronary heart disease (17.0%), etc.[9] The difference of demographic profile in this study and those in Turkey as well as the similarity of those in this study with another study in Indonesia may be explained by the difference in the prevalence of risk factors of stroke across countries. In addition, the similarities of demographic profile among groups in this study may reduce the confounding factors from another vascular risk factors to determine the independent effect of hypoplasia in clinical symptoms.

However, compared with the general demographic profile in Indonesia, this study had more cases of previous stroke, especially in the VAH (77.1%) and ACAH (68.2%) groups compared with PCAH group (44.4%). The lower prevalence of previous stroke in PCAH group may be associated with the lower number of subjects in that group. Albeit statistically insignificant among hypoplasia groups, the increased prevalence of recurrent symptoms in the cerebral artery hypoplasia group described in this study was interesting to be further studied.

Observing the association between hypoplasia and clinical symptoms, the clinical symptoms of hypoplasia were mostly stroke (89.4%), of which 25.4% presented with posterior circulation symptoms. The VAH and PCAH subjects had a higher percentage of posterior circulation symptoms (28.1% and 50.0%, respectively) than ACAH (14.3%), albeit insignificant. The symptoms of 59 subjects with stroke in this study were hemiparesis and cranial nerve palsy [44 (74.6%) subjects] as well as vertigo [15 (25.4%) subjects]. While stroke remains the most common primary diagnosis in this study, we found seven subjects with nonstroke symptoms in this study, including headache [6 (85.7%) subjects] and epilepsy [1 (14.3%) subjects].

A study from Stevano et al in Indonesia with 153 subjects of stroke/TIA provided that the overall prevalence of posterior circulation symptoms in stroke was 20.3%.[9] Therefore, there were increased number of posterior circulation symptoms in the posterior cerebral vessel hypoplasia compared with those in ACAH or in general population of stroke.

Another study by Bakalarz et al described that the most common symptoms of VAH in decreasing order were limb paresis (47.5%), cranial nerve palsy (43.8%), vertigo (27.5%), decreased consciousness (17.5%), aphasia (23.8%), sensory disturbance (13.8%), headache (7.5%), visual field disturbance (6.3%), ataxia (3.8%), and monocular vision (3.8%). The most common primary diagnosis in symptomatic VAH were ischemic stroke (41%), transient ischemic attack, cranial nerve disorders (8.8%), epilepsy (5.0%), and demyelinating disease (5.0%).[10] In conclusion, there were similar profile of clinical characteristics between this study and previous studies. Interestingly, while posterior circulation symptoms of vertigo, limb paresis, or cranial nerve palsy were predominant in cerebral artery hypoplasia (especially VAH), they were not limited to posterior circulation. It was thought that hypoplasia may not only affect respective distal vascular territory, but also the overall cerebral blood flow. A study regarding the cerebral blood flow changes in VAH described that VAH was associated with lower internal carotid artery (anterior circulation) blood flow in older subjects whereas non-VAH was associated with lower basilar artery (posterior circulation) blood flow in older subjects.[11] The study of cerebral blood flow in cerebral artery hypoplasia was beyond the scope of this study.

Determining the association between hypoplasia and brain MRI ischemic lesions, there were 91.4%, 55.6%, and 86.4% cases of brain MRI ischemic lesions in VAH, PCAH, and ACAH subjects, respectively. The fourth power of vessel radius was associated with the flow rate based on Poiseuille's law. Therefore, cerebral artery hypoplasia may be associated with reduced blood flow, which may play an important role in the ischemia process.[12] The different number of brain MRI ischemic lesions among groups may be associated with some variations in the circle of Willis and the smaller subject size in PCAH groups.[8]

There were no differences between all demographics and clinical parameters and the presence of symptomatic complaints and brain MRI lesions among VAH, PCAH, and ACAH subjects as well as the general profile of stroke, except in the variables of previous history of stroke. It can be concluded that cerebral artery hypoplasia may be considered as an additional risk factor to the conventional vascular risk factors for subtle brain ischemia, symptomatic complaints, and recurrent stroke.[3,4,12]

Cerebral artery hypoplasia was hypothesized either independently or in association with cardiovascular risk factors to produce clinical symptoms. Decreased vessel lumen may independently produce regional hypoperfusion. This pathomechanism may be countered with the presence of vessel collaterals, which were also heterogeneous within every individual.[13] In respect to cardiovascular risk factors, decreased vessel lumen may potentiate slow flow and high sheer stress locally, which increases the susceptibility of atherosclerosis, stenosis, or occlusion, and the risk of ischemic stroke due to atherosclerosis at the respective region, as well as disturbance in general cerebral blood flow at anterior or posterior circulation.[11,14]

The role of cerebral artery hypoplasia in symptomatic clinical manifestations had largely been observed in VAH, which is a common congenital anatomical variation. However, it may be observed asymptomatically in healthy individuals as well.[11] This study also described that there were no differences in the age of onset of symptomatic clinical manifestations in cerebral artery hypoplasia and general stroke population. Therefore, it was thought that cerebral artery hypoplasia acted more as a predisposing factor for other cardiovascular risk factors rather than a single independent risk factor, for symptomatic clinical manifestations.

Another finding in this study was the similarities of every clinicodemographic profile of cerebral artery hypoplasia in respect to the occurrence of clinical symptomatic manifestations. While VAH was considered as the most frequent anatomical variant, we suggest that the presence of other types of hypoplasia should also be considered as a potential aggravating risk factor for clinical symptomatic manifestation.[15] More studies were needed to justify this statement.

The strength of this study included the use of the gold standard of DSA as a measure for determining hypoplasia. Brain MRI was also performed on all subjects to assess covert brain ischemia. However, some limitations in this study included the small sample size of 66 subjects although total sampling has been performed during the period of the study. Comparison between hypoplasia and non-hypoplasia subjects was also not

conducted. This limitation was countered by external comparison with the general profile of stroke. Flow and stenosis assessment, which may present as the effect of hypoplasia, were also beyond the scope of this study. Further studies comparing hypoplasia and non-hypoplasia subjects with the additional focus on flow and stenosis assessment were valuable in deeper understanding of the impact of hypoplasia. This may potentially lead to the change in the paradigm of hypoplasia management in stroke prevention. Despite its limitation, this study supported most recent studies regarding cerebral artery hypoplasia as the potential adjunctive risk factor for clinical symptoms including stroke and posterior circulation symptoms. While studies regarding VAH and stroke were the most abundant, PCAH and ACAH should also be considered as potential risk factors.

CONCLUSION

The prevalence of cerebral artery hypoplasia was 8.6%. There were no differences in the demographic, clinical, and brain MRI ischemic profile among hypoplasia subjects of VAH, PCAH, and ACAH, to the general profile of stroke in Indonesia. The number of strokes was similar among VAH, PCAH, and ACAH, with clinical symptoms tended to correspond to the involving artery hypoplasia. In addition, recurrent stroke was also observed to be higher in hypoplasia subjects than in the general stroke population. Further studies are required to support this finding.

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REFERENCES

1. Ministry of Health of the Republic of Indonesia. National Riskesdas Report 2018. Health Research and Development Agency; 2018:674.
2. Iqbal S. A comprehensive study of the anatomical variations of the circle of Willis in adult human brains. J Clin Diagn Res. 2013;7(11):2423-7.
3. Erdal Y, Bayramoğlu B, Şişman Ç, Mahmutoğlu AS, Emre U. The relationship between vertebral artery hypoplasia and posterior circulation stroke. Istanbul Med J. 2022;23(1):16-9.
4. Bae JH, Ryu JC, Ha SH, Kim BJ, Kang DW, Kwon SU, et al. Association of left vertebral artery hypoplasia with posterior circulation stroke and the functional outcome of patients with atrial fibrillation-related cardioembolic stroke. Am J Neuroradiol. 2023;44(1):65-9.
5. Lakhotia M, Pahadiya HR, Prajapati GR, Choudary A, Gandhi R, Jangit H. A case of anterior cerebral artery A1 segment hypoplasia syndrome presenting with right lower limb monoplegia, abulia, and urinary incontinence. J Neurosci Rural Pr. 2016;7(1):189-91.
6. Chuang Y, Liu C, Pan P, Lin C. Anterior cerebral artery A1 segment hypoplasia may contribute to A1 hypoplasia syndrome. Eur Neurol. 2007;57:208-11.
7. Rinaldo L, Mccutcheon BA, Murphy ME, Bydon M, Rabinstein AA, Lanzino G. Relationship of A1 segment hypoplasia to anterior communicating artery aneurysm morphology and risk factors for aneurysm formation. J Neurosurg. 2017;127(July):89-95.
8. Dinc Y, Ozpar R, Emir B, Hakyemez B, Bakar M. Vertebral artery hypoplasia as an independent risk factor of posterior circulation atherosclerosis and ischemic stroke. Medicine. 2021;100:38.
9. Stevano R, Margono JT, Sutanto A. Clinical profile and risk factors of stroke: a comparative analytical study between young and old onset. Magna Neurol. 2023;1(1):13-6.
10. Bakalarz M, Ro JJ. Clinical characteristics of patients with vertebral artery hypoplasia as a full Wallenberg syndrome. Int J Environ Res Public Health. 2022;19:9317.
11. Miller KB, Gallo SJ, Rivera-Rivera LA, Corkery AT, Howery AJ, Johnson SC, et al. Aging brain vertebral artery hypoplasia influences age-related differences in blood flow of the large intracranial arteries. Aging Brain. 2021;1:100019.
12. Oh HG, Lee SC. Clinical significance of vertebral artery hypoplasia in lateral medullary infarction. Soonchunhyang Med Sci. 2017;23(2):104-7.
13. Davidoiu A. The fetal type of posterior cerebral artery. Medicina (B Aires). 2023;59:231.
14. Gaigalaite V, Vilimas A, Ozeraitiene V, Dementaviciene J, Janilionis R, Kalibatiene D, et al. Association between vertebral artery hypoplasia and posterior circulation stroke. BMC Neurol. 2016;16(1):1.
15. Bhanu SP, Pentyala S, Sankar DK. Incidence of hypoplastic posterior communicating artery and fetal posterior cerebral artery in Andhra population of India: a retrospective 3-Tesla magnetic resonance angiographic study. Anat Cell Biol. 2020;53:272-8.

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