

Anatomical Profile of Vertebrobasilar System Based on Angiographic Studies

Gilbert Tangkudung¹✉, Finny Warouw¹, Kennytha Yoesdyanto², Vinson Hartoyo¹

¹ Department of Neurology, Faculty of Medicine, Universitas Sam Ratulangi; Prof. dr. R. D. Kandou Hospital, Manado, Indonesia

² Department of Neurology, Faculty of Medicine, Universitas Pelita Harapan, Tangerang, Indonesia

Corresponding Author:

Gilbert Tangkudung

Department of Neurology, Faculty of
Medicine, Universitas Sam Ratulangi; Prof.
dr. R. D. Kandou Hospital, Manado,
Indonesia

Email: gilbert.tangkudung@gmail.com

Introduction: Variations of cerebral vasculature in vertebrobasilar system (VBS) occur during embryogenesis and may contribute to cerebrovascular events. These anatomical variants occur in approximately 7% of cases with a 91% prevalence noted in the symmetrical caudal fusion variant. Cerebral angiography remains the most sensitive method for diagnosing these anomalies. **Objective:** This study aims to identify angiographic variations in the VBS. **Method:** This descriptive observational study used a cross-sectional approach, analyzing secondary data from stroke patients who underwent angiographic procedures between December 2017 and August 2020 at Prof. dr. R. D. Kandou Hospital, Manado. The sample size included the total population of stroke patients who met the inclusion criteria. Data were processed using SPSS version 32. **Result:** A total of 277 samples were analyzed. The most commonly observed variations were symmetric cranial fusion (81.50%) and asymmetric caudal fusion (15.42%). One case of symmetric caudal fusion had a basilar tip aneurysm. While most patients exhibited normal anatomy, variants of the posterior cerebral artery (PCA) were also observed, including absence (6.17%) and hypoplasia (7.48%). The superior cerebellar artery (SCA) was absent in 1.76% of cases. Absence of the anterior inferior cerebellar artery (AICA) and posterior inferior cerebellar artery (PICA) was observed in 13.56% of cases; however, these were compensated by complex vascular formations involving collateral sources (21.58%). **Conclusion:** This study concludes that anatomical variations in cerebral vasculature, including perforators and anastomoses, can be effectively observed through angiographic studies. Careful patient selection is essential to rule out secondary causes of hemifacial spasm and to identify underlying neurovascular contacts.

Keywords: Angiography, Duplication, Fenestration, Hypoplasia, Vertebrobasilar system

Received: March 13, 2025

Revised: April 22, 2025

Accepted: April 30, 2025

Published: May 28, 2025

Highlights

- Anatomical variations in blood vessels can significantly affect circulatory hemodynamics
- These variants also pose a risk of developing intracranial pathologies such as aneurysms
- Early detection can help prevent further complications

Introduction

Human intracranial circulation anatomically is divided into two main systems: the anterior and posterior circulations. The vertebrobasilar system (VBS) is part of the

intracranial posterior circulation and consists of vertebral and basilar arteries which supply blood to the brainstem, spinal cord, cerebellum, thalamus, and occipital lobe.^{1,2,3}



Disturbances in the vertebrobasilar system can lead to neurological deficits; therefore, a comprehensive understanding of its anatomy and clinical significance is pivotal for identifying certain syndromes and for treatment planning.^{4,5}

Variations in vertebrobasilar blood vessels occur during embryogenesis. These variations can contribute to cerebrovascular events, particularly aneurysms, which can be fatal if ruptured and result in bleeding into the posterior fossa.^{6,7} The incidence of basilar aneurysms is approximately 7% with a 91% prevalence observed in the symmetric caudal fusion variant. In other variants, the presence of additional perforator branches or anastomoses may serve to compensate the altered circulation.^{8,9}

Digital subtraction angiography (DSA) remains the gold standard diagnostic tool for detecting vascular variants. Angiography allows visualization of the origin of blood vessels and provides precise information regarding vessel absence and compensatory vascular pathways.^{3,10}

Objective

This study aims to provide an angiographic profile of VBS.

Method

This observational descriptive study employed a cross sectional approach using secondary data from patients who underwent angiographic procedures between December 2017 and August 2020 at Prof. dr. R. D. Kandou Hospital, Manado. This study was approved by the Ethical Committee of the Indonesian Ministry of Health Research and Health Development (Ethical Approval No. 093/EC/KEPK-KANDOU/X/2020).

Inclusion criteria were patients with signs and symptoms indicative of a cerebrovascular event (stroke) who had undergone angiographic evaluation. Exclusion criteria included patients diagnosed with intracranial pathologies of non-vascular origin. All data were recorded and analyzed accordingly.

The data obtained were analyzed using SPSS version 32. Vessels were categorized according to their embryologic morphology in the vertebrobasilar system, specifically: the posterior cerebral artery (PCA), superior cerebellar artery (SCA), anterior inferior cerebellar artery (AICA), posterior inferior cerebellar artery (PICA), basilar artery (BA) and vertebral artery (VA).

Result

A total of 227 subjects underwent angiographic evaluation between December 2017 and August 2020. Clinically significant variant identified included symmetric cranial fusion (81.5%), asymmetric caudal fusion (15.42%) and symmetric caudal fusion (3.08%) (Figure 1).

Variants observed in the PCA (table 1) included bilaterally absent vessels (0.88%), bilateral hypoplasia (0.44%), and unilateral hypoplasia on the left (3.08%) and right (2.21%) sides. The majority of PCA configurations were normal (86.34%).

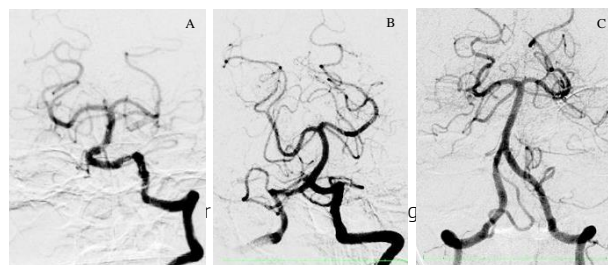


Figure 1. Fusion variants in vertebrobasilar system: A. Symmetric cranial fusion, B. Symmetric caudal fusion, C. Asymmetric caudal fusion.

Bilateral duplications of the SCA were found in 1.76% of cases. Among these, two cases showed origins from different vessels (the BA and PCA respectively), while the remaining three cases originated from the BA.

In the left circulation, variants included absent SCA (0.88%) and duplications (10.57%). The duplicated SCAs originated from the PCA (16.67%), the BA (70.83%) and both the PCA and BA (12.5%). One case of triplication (0.44%) was also noted. Among cases with single SCA, variants with unilateral origin from the PCA were found in both the left (2.21%) and right (2.21%) circulations, corresponding to the asymmetric caudal fusion category (Table 2).

In the right circulation, SCA variants included absence (0.88%) and duplication (4.40%). Among duplicated vessels, 10% originated from both the PCA and BA, and 90% from the BA alone. One case of triplication (0.88%) originated entirely from BA. Symmetric fusion was classified based on the origin of both SCAs from the same vessel—cranial symmetric fusion if from the BA (73.56%) and caudal symmetric fusion if from the PCA (2.21%) (Table 3).

Table 1. Variants of PCA

Variants	Counts (n)	Percentage (%)
Bilateral absence	2	0.88%
Bilateral hypoplastic	1	0.44%
Left-sided absence	7	3.08%
Left hypoplastic	8	3.52%
Right-sided absence	5	2.21%
Right hypoplastic	8	3.52%
Normal	196	86.35%
Total	227	100%

Table 2. Variants of SCA

Variants	Counts (n)	Percentage (%)
Bilateral duplication	4	1.76%
Bilateral BA origin	167	73.56%
Bilateral PCA origin	5	2.21%

Table 2 (continued). Variants of SCA

Variants	Counts (n)	Percentage (%)
Left-sided absence	5	0.88%
Left-sided duplication	2	10.57%
Left PCA origin	24	2.21%
Left-sided triplication	5	0.44%
Right-sided absence	1	0.88%
Right-sided duplication	2	4.40%
Right PCA origin	10	2.21%
Right-sided triplication	5	0.88%
Total	227	100%

Table 3. Variants of vertebrobasilar system

Variants	Counts (n)	Percentage (%)
Symmetric cranial fusion	185	81.50%
Symmetric caudal fusion	7	3.08%
Asymmetric caudal fusion	35	15.42%
Total	227	100%

AICA variants included bilateral absence (1.32%), bilateral duplication (0.44%), left-sided absence (1.76%), and right-sided absence (4.40%). In some cases, where AICA was not visualized, collateral supply was provided by either PICA (PICA-AICA complex, 1.76%) or the SCA (SCA-AICA complex, 1.76%). These complexes were observed in cases of bilateral AICA absence, with proportions of 64% for PICA-AICA and 33% for SCA-AICA (Table 4) (Figure 2). The remaining cases were considered normal (85.47%).

Table 4. Variants of AICA

Variants	Counts (n)	Percentage (%)
Bilateral absence	3	1.32%
Bilateral duplication	1	0.44%
Left-sided absence	4	1.76%
Right-sided absence	10	4.40%
PICA-AICA complex	11	4.85%
SCA-AICA complex	4	1.76%
Normal	194	85.47%
Bilateral absence	3	1.32%
Total	227	100%

Table 5. Variants of PICA

Variants	Counts (n)	Percentage (%)
Bilateral AICA-PICA complex	8	3.52%
Bilateral absence	1	0.44%
Left-sided absence	5	2.21%
Left AICA-PICA complex	13	5.73%
Right-sided absence	8	3.52%
Right AICA-PICA complex	4	1.76%
Total	227	100%

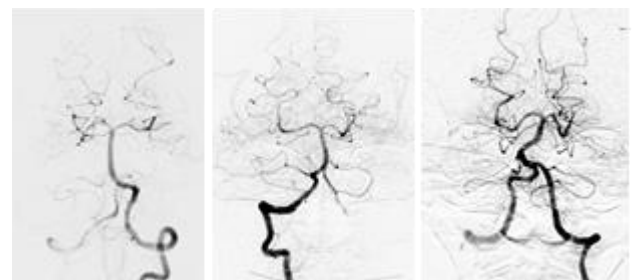
PICA variants included bilateral absence (0.44%), unilateral left-sided absence (2.21%), and right-sided absence (3.52%). AICA-PICA complexes were identified both bilaterally (3.52% in cases of bilateral PICA absence)

and unilaterally (5.73% on the left, 4.40% on the right). The majority of PICAs were normal (80.18%) (Table 5).

Table 6. Variants of vertebral artery

Variants	Counts (n)	Percentage (%)
Left-sided absence	1	0.44%
Left hypoplastic	1	0.44%
Right hypoplastic	8	3.52%
Normal	217	95.60%
Total	227	100%

VA variants included left-sided absence (0.44%) and hypoplasia (right-sided 3.52%, left-sided 0.44%). All other cases (95.60%) were normal. No significant morphological variants were identified in the BA (Table 6).

**Figure 2.** Variants found in angiographic study A. Right AICA-PICA complex and VA hypoplastic, B. Right PICA-AICA complex, C. Right SCA triplication.

Discussion

The cerebral posterior circulation consists of two vertebral arteries and one basilar artery, which together form the vertebrobasilar system and contribute approximately 20% of brain's blood supply. These arteries arise from the subclavian arteries, ascend through the cervical region, enter the cranium, and branch into smaller vessels.⁶ These smaller arteries merge to form the basilar artery, which then bifurcates into PCAs. Just proximal to this bifurcation, the BA also gives rise to SCAs, which supply the lateral aspect of the pons midbrain, as well as the superior region of the cerebellum.^{11,12,13}

Embryological studies have classified the morphology of the basilar apex into three categories: symmetric cranial fusion, symmetric caudal fusion, and asymmetric caudal fusion. Angiographic classification of the basilar apex is primarily based on the origin of the SCAs. In cranial fusion, the SCAs arise directly from the basilar artery. In caudal fusion, they originate from the P1 segment of the PCA. Caudal fusion can be further divided into symmetric and asymmetric types. The asymmetric fusion is defined by a discrepancy in the origin of the SCAs—for example, one SCA arising from the PCA while the other from BA. Meanwhile, the symmetric caudal fusion variant is associated with an increased risk of aneurysm formation at the BA apex due to higher blood pressure accumulation at this endpoint. Similarly, the asymmetric caudal fusion variant also poses a risk of aneurysm development, likely due to increased friction caused by altered blood flow

dynamics. This friction, a result of anatomical variations in vessel structure, can significantly affect blood pressure within the BA.^{10,12,14} Our study identified one case of a basilar artery aneurysm in a patient with symmetric caudal fusion, supporting the existing literature that implicates both high pressure and friction in aneurysm development.

Perforator arteries can be found to be missing in some cases. Most perforator arteries developed from main arteries on counterpart side. Thus, one group of perforator arteries can supply both areas of different brain hemispheres. In other instances, blood supply is assisted by ipsilateral branch of adjacent blood vessels.^{8,9,13} Others can also connected through anastomoses, however its significance is negligible especially in cases of perforator occlusion. In terms of perforator, embryologically, the absence of AICA and PICA can be compensated through formation of complexes. For example, absence of AICA will results in formation of AICA-PICA complex or SCA-AICA complex to compensate vascularization territory.^{15,16} This compensatory complex formation is also found in our study.

Hypoplastic arteries are among the most frequently observed variants in adults. Although their significance has not been clearly established in a population-based study, several post-mortem case studies suggest an association between hypoplasia and cerebral infarction. However, this theory remains controversial due to the generally intact lumen structure of hypoplastic vessels.^{17–20} Similarly, intracranial arterial fenestration and duplication have been linked to the formation of saccular aneurysms. The widely accepted hypothesis is that the proximal and distal ends of duplicated vessels have reduced smooth muscle and collagen content, making them more susceptible to rupture. This is supported by findings of subarachnoid hemorrhage in patients with duplicated arteries and no identified risk factors.^{21–24} While the clinical significance of many anatomical variants in this study remains unclear, increasing awareness toward these variations is important, particularly in cases of atypical stroke.

Our study benefited from a considerable sample size; however, it is limited by the inability to control for other vascular-related risk factors due to its retrospective design. Additionally, this study design did not allow for follow-up data collection to evaluate the recurrence of neurological events.

Conclusion

This study identified various intracranial arterial variants, particularly within the VBS. Some of these variants can significantly impact blood flow hemodynamics. Angiography, as the gold standard imaging technique, proved valuable in visualizing both anatomical variations and associated perforators. Further prospective studies with larger sample sizes are needed to enhance our understanding of these vascular variations and their clinical implications.

Acknowledgement

We would like to express our sincere gratitude to Prof. dr. R. D. Kandou Hospital for all necessary supports given during our research period.

Conflict of Interest

All authors declare no conflict of interest.

Ethic consideration

This study was approved by the Ethical Committee of the Indonesian Ministry of Health research and Health Development with ethical approval number 093/EC/KEPK-KANDOU/X/2020.

Funding

None

Author contribution

Gilbert Tangkudung: Conceptualization, data curation, investigation, methodology, resources, supervision, validation, writing—original draft, writing—review, and editing. **Finny Warouw:** Conceptualization, data curation, methodology, supervision, validation, writing—review and editing. **Kennytha Yoesdyanto:** Data curation, investigation, methodology, project administration, writing—original draft, writing—review and editing. **Vinson Hartoyo:** Methodology, project administration, resources, software, writing—review and editing.

References

1. Agarwal N, Carare RO. Cerebral vessels: an overview of anatomy, physiology, and role in the drainage of fluids and solutes. *Front Neurol.* 2021;11:611485. DOI: [10.3389/fneur.2020.611485](https://doi.org/10.3389/fneur.2020.611485)
2. Srichawla BS, Garcia-Dominguez MA. Regional dynamic cerebral autoregulation across anterior and posterior circulatory territories: a detailed exploration and its clinical implications. *World J Crit Care Med.* 2024;13(4):1–10. DOI: [10.5492/wjccm.v13.i4.97149](https://doi.org/10.5492/wjccm.v13.i4.97149)
3. Govindarajan M, Vijayeswaran N. Anatomical variations and its clinical significance in vertebrobasilar system – a cadaveric study in the Indian population. *Int J of Adv Res.* 2018;6:319–24. DOI: [10.21474/IJAR01/7678](https://doi.org/10.21474/IJAR01/7678)
4. Ballout AA, Libman RB, Schneider JR, Black K, Sideras P, Wang JJ, et al. Vertebrobasilar stroke: association between infarction patterns and quantitative magnetic resonance angiography flow state. *J Am Heart Assoc.* 2022;11:e023991. DOI: [10.1161/JAHA.121.023991](https://doi.org/10.1161/JAHA.121.023991)
5. Sudharmadji S, Leslie GM, Herryanto D. Analysis of the relationship between symptoms, clinical diagnosis, and findings of CT-scan images to pictures of vertebrobasilar artery insufficiency. *Asian J Health Sci.* 2024;3(10):265–77. DOI: [10.58631/ajhs.v3i10.127](https://doi.org/10.58631/ajhs.v3i10.127)

6. Bonasia S, Di Caterino F, Robert T. Embryology of the vertebral artery and variants of the adult. *Neurochirurgie*. 2024;70(3):101517. DOI: [10.1016/j.neuchi.2023.101517](https://doi.org/10.1016/j.neuchi.2023.101517)
7. Matsukawa H, Kamiyama H, Noda K, Ota N, Takahashi O, Shonai T, et al. Embryological basilar apex disposition as a risk factor of basilar apex aneurysm. *J Clin Neurosci*. 2018;58:79–82. <https://doi.org/10.1016/j.jocn.2018.10.004>
8. Djulejić V, Marinković S, Milić V, Georgievski B, Rašić M, Aksić M, et al. Common features of the cerebral perforating arteries and their clinical significance. *Acta Neurochir*. 2015;157:743–54. DOI: [10.1007/s00701-015-2378-8](https://doi.org/10.1007/s00701-015-2378-8)
9. Antunović V, Mirčić A, Marinković S, Brigante L, Mališ M, Georgievski B, et al. Clinical significance of the cerebral perforating arteries. *PRILOZI*. 2017;38(3):19–29. DOI: [10.2478/prilozi-2018-0003](https://doi.org/10.2478/prilozi-2018-0003)
10. Aburto-Murrieta Y, Marquez-Romero JM, Martínez-Arellano P, Serrano-Arias FE, Montenegro-Rosales HA, López-Mena D. Anatomical variations of the intracranial arteries and their association with intracranial aneurysms: insights from digital subtraction angiographies. *Neuroradiol J*. 2025. DOI: [10.1177/19714009251313516](https://doi.org/10.1177/19714009251313516)
11. Reyes-Soto G, Pérez-Cruz JC, Delgado-Reyes L, Castillo-Rangel C, Cacho Diaz B, Chmutin G, et al. The vertebrobasilar trunk and its anatomical variants: a microsurgical anatomical study. *Diagnostics*. 2024;14(5):534. DOI: [10.3390/diagnostics14050534](https://doi.org/10.3390/diagnostics14050534)
12. Ota T. Functional arterial anatomy of the brain. *Stroke Vasc Interv Neurol*. 2022;2(5):e000446. DOI: [10.1161/SVIN.122.000446](https://doi.org/10.1161/SVIN.122.000446)
13. Vogels V, Dammers R, Van Bilsen M, Volovici V. Deep cerebral perforators: anatomical distribution and clinical symptoms: an overview. *Stroke*. 2021;52(10):e660–74. DOI: [10.1161/STROKEAHA.120.034096](https://doi.org/10.1161/STROKEAHA.120.034096)
14. Park H, Son YJ, Hong N, Kim SB. The relationship between non-aneurysmal spontaneous subarachnoid hemorrhage and basilar tip anatomy. *J Cerebrovasc Endovasc Neurosurg*. 2022;24(3):232–40. DOI: [10.7461/jcen.2022.E2022.01.004](https://doi.org/10.7461/jcen.2022.E2022.01.004)
15. Błaszczuk M, Ochwat K, Necka S, Kwiecińska M, Ostrowski P, Bonczar M, et al. The arterial anatomy of the cerebellum—a comprehensive Review. *Brain Sci*. 2024;14(8):763. DOI: [10.3390/brainsci14080763](https://doi.org/10.3390/brainsci14080763)
16. Hou K, Xu K, Yu J. Endovascular treatment of anterior inferior cerebellar artery trunk aneurysms. *Interv Neuroradiol*. 2022;28(5):604–12. DOI: [10.1177/15910199211049054](https://doi.org/10.1177/15910199211049054)
17. Bakalarz M, Roźniecki JJ, Stasiotek M. Clinical characteristics of patients with vertebral artery hypoplasia. *Int J Environ Res Public Health*. 2022;19(15):9317. DOI: [10.3390/ijerph19159317](https://doi.org/10.3390/ijerph19159317)
18. Göktürk N, Kara T, Tural İÇ, Yüksel M, Yurttutan N. Vertebrobasilar system variations; analysis with multi-detector CT angiography. *Med Sci Discov*. 2024;11(7):199–204. DOI: [10.36472/msd.v11i7.1159](https://doi.org/10.36472/msd.v11i7.1159)
19. Dinç Y, Özpar 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):e27377. DOI: [10.1097/MD.00000000000027280](https://doi.org/10.1097/MD.00000000000027280)
20. Hsu CF, Chen KW, Su CH, Shen CY, Chi HY. Bilateral vertebral artery hypoplasia and fetal-type variants of the posterior cerebral artery in acute ischemic stroke. *Front Neurol*. 2021;12:582149. DOI: [10.3389/fneur.2021.582149](https://doi.org/10.3389/fneur.2021.582149)
21. Dong J, Mei Y, Bai X, Tong X, Dmytriw AA, Yang B, et al. Hemodynamic differences between basilar artery fenestration and normal vertebrobasilar artery: a pilot study. *Front Neurol*. 2022;12:766174. DOI: [10.3389/fneur.2021.766174](https://doi.org/10.3389/fneur.2021.766174)
22. Omotoso BR, Harrichandparsing R, Moodley IG, Satyapal KS, Lazarus L. Fenestration of the vertebrobasilar junction detected with multidetector computed tomography angiography. *Folia Morphol*. 2022;81(2):510–4. DOI: [10.5603/FM.a2021.0028](https://doi.org/10.5603/FM.a2021.0028)
23. Zhu DY, Fang YB, Wu YN, Li Q, Duan GL, Liu JM, et al. Treatment of fenestrated vertebrobasilar junction-related aneurysms with endovascular techniques. *J Clin Neurosci*. 2016;28:112–6. DOI: [10.1016/j.jocn.2015.09.018](https://doi.org/10.1016/j.jocn.2015.09.018)
24. Kritikos M, Sharma N, Sedora-Roman N, Choudhri O, Pukenas B, Hurst R, et al. Duplication of the vertebral artery: A series of four cases. *Interv Neuroradiol*. 2018;24(6):596–600. DOI: [10.1177/1591019918783973](https://doi.org/10.1177/1591019918783973)