

Interesting basis of vertebrobasilar arterial territory

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Keywords

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Abstract

Background: Vertebrobasilar arterial territory nourishes one-quarter of human brain. It constitutes some vital and strategic parts of the central nervous system.

Methods: A number of keywords (vertebral, basilar, artery, and territory) were searched in MEDLINE (Ovid and PubMed) as well as Google, ProQuest, Scopus, Cochrane Library, and Science Direct online databases. Only articles containing all keywords were included. We also reviewed archives of libraries in Mashhad University of Medical Sciences (Iran) for all anatomy, embryology, neurology, and neuroscience books and journals about vertebrobasilar arterial territories.

Results: The vertebrobasilar arterial (VA) system has a high incidence of variations, anomalies, and persistent fetal vessels. Two important anatomic facts explain why VA origin lesions seldom cause chronic hemodynamically significant low flow to the vertebrobasilar system. First, the VAs are paired vessels that unite to form a single basilar artery. Second, the extracranial VA gives off numerous muscular and other branches as it ascends in the neck. Thus, in the VA system, there is much more potential for development of adequate collateral circulation. Even when there is bilateral occlusion of the VAs at their origins, patients do not often develop posterior circulation infarcts.

Conclusion: VA origin disease is more benign than ICA origin disease from hemodynamic aspect. This important point could make influence in therapeutic interventional decisions in asymptomatic VA origin stenosis.

The vertebral arteries (VA) are usually the first branches of the subclavian arteries. In 5% of persons, the left VA arises directly from the arch of the aorta between the common carotid and the left subclavian artery. In this case, the left VA would not fill from a left brachial injection.¹ Rarely, the right VA arises as a separate branch from innominate artery and not from the subclavian artery.² One of the vertebral arteries is often larger than the other.¹ When one is smaller, the other is likely to be unusually large, so that the amount of blood supplied to the posterior circulation remains constant.^{1,2} In 45% of people, the left VA is larger, in 21%, the right VA is larger, and in 24%, the arteries are of equal size.³ The average inner diameter of the VA is 3.5 mm (1.5-5 mm).⁴ In 10% of cases, one VA is so hypoplastic (diameter < 2 mm) that it carries very little blood and in rare cases, one VA is absent altogether.⁵ The left or right VA may Sometimes have an anomalous origin (e.g. duplicated origin).⁵

Traditionally, the VA is divided into four segments. In the first segment, the artery courses directly cephalad from its origin as the first branch of the subclavian artery to enter the costotransverse foramen of the fifth or sixth cervical vertebra. The second segment of VA then travels through the bony tunnel formed by the adjacent transverse processes and the ligaments of the sixth cervical vertebra, up to the first cervical vertebra (the atlas).^{5,6} In this tunnel, it is accompanied by veins and a very dense plexus of sympathetic nerves that interconnect with cervical ganglia.^{5,7} The third segment is highly tortuous: the VA emerges from the transverse foramen of the second cervical vertebra and courses posteriorly and

laterally toward the costotransverse foramen of the atlas.^{5,7} It circles the posterior arch of the first cervical vertebra and passes between the atlas and occiput within the suboccipital triangle where it is covered only by soft tissues. It forms a loop akin to that of carotid siphon and anastomoses freely with the occipital branch of external carotid artery by its muscular branches.⁵ Entering the skull through the foramen magnum, the fourth segment of two VAs pierces the dura mater and ascend along the ventrolateral medulla oblongata. The two VAs usually merge at the level of pontomedullary junction to form the basilar artery.¹

The course of the BA is also variable. It runs straight on the midline of the pons in half of people and displays tortuosity in the other half.¹ However, it constantly ends between peduncles of the mesencephalon. The average length of BA is 30 mm (21-41 mm) and its average inner diameter is 3 mm (2.5-3.5 mm).⁴ Just after piercing the dura, each VA gives off its anterior spinal ramus.^{1,3} The posterior inferior cerebellar artery (PICA) usually arises from the VA an average of 8.5 mm above the foramen magnum.⁴ Occasionally, the PICA arises extracranially and courses cephalad within the spinal canal or originates from the ascending pharyngeal artery.^{8,9} The VA may terminate in the PICA. In this case, the distal segment is hypoplastic or nonexistent and the VA is small compared to the contralateral side.^{8,9} The medial branch of PICA may also arise directly from the VA with the lateral branch arising from the BA or more commonly from the anterior inferior cerebellar artery (AICA).^{3,5} One PICA is entirely lacking in 15% of individuals and is hypoplastic in 5%.^{3,5} The PICA encircles the medulla, divides into two main lateral and medial branches, and supplies the suboccipital surface in the caudal part of the cerebellum.^{1,10} The AICA arises from the caudal third of the BA in 75%. It sometimes arises from the middle third and is lacking in 4% of individuals.¹ However, it can arise from the VA or BA by a common trunk together with the PICA. Multiple ipsilateral AICAs are observed in 12% of individuals.^{3,11} The AICA encircles the lower pons and supplies a small area of the anterior and medial cerebellum.¹ The PICA and AICA are often reciprocally related in size, i.e. in the case of a large AICA, the PICA is small.¹²

The superior cerebellar artery (SCA) arises from the rostral BA just before its bifurcation at the level of interpeduncular cistern in 80% of people. In the remaining 20%, the SCA arises from the mesencephalic artery, or P1 segment of the posterior cerebral artery (PCA). The SCAs are occasionally duplicated.^{5,13} Each SCA has a short trunk and two main medial and lateral branches which supply the

rostral half of the cerebellar hemisphere and dentate nucleus.^{5,13} The PICA, AICA, SCA, and their branches are connected by numerous free anastomoses which limit infarct size in patients who have PICA, AICA, SCA, VA, or BA occlusions.^{5,14} Many leptomeningeal anastomoses exist between the three cerebellar arteries on the same site, but they can also be present between the PICA and SCA on both sides.^{5,14} These leptomeningeal anastomoses also make the exact site and extent of cerebellar infarct quite unpredictable.^{3,15}

The major branches of BA are generally uniform. The most common variation is the internal auditory artery (usually an AICA branch) which may arise directly from the BA.³ During early fetal life, the internal carotid artery supplies the posterior hemispheres and brainstem via posterior communicating arteries.^{3,16} In one third of people, this primitive vascular pattern persists and the connecting segment from the BA to the PCA (called the basilar communicating artery, mesencephalic artery, or P1 segment of the PCA) remains vestigial.³ When the PCA originates from the ICA, the supply area of mesencephalic artery is restricted to the upper brain stem. In such conditions, the PCA may fill from carotid injection and not after VA opacification.¹⁷ In 2% of humans, this primitive circulatory pattern is bilateral.^{3,5,16} Even more rarely, the BA may be hypoplastic in its distal segment and end in the SCAs. The other primitive connection which remains in 0.2% of adults is the trigeminal artery.^{3,18} The trigeminal artery arises from the ICA as it enters the cavernous sinus proximal to the carotid siphon and penetrates the sella turcica or the dura near the clivus to join the BA between the AICA and SCA branches.^{2,19} Therefore, in some individuals with a dominant posterior communicating artery (PCoA) or a persistent trigeminal artery, embolism from the carotid territory can cause occlusion of the PCA and even the brainstem.

Persistence of the hypoglossal artery is the next most common variant.^{2,3,5} This vessel originates from the ICA in the neck, usually between the first and third first cervical vertebrae, and courses posteriorly to enter the hypoglossal canal from which it joins the BA.^{2,20} In these cases, the VAs or BA are small or hypoplastic.^{2,3} In contrast to the variability of the vertebrobasilar arteries and their branches, the intramedullary territories of the brain stem are remarkably constant.³ There are three groups of arterial penetrators origin from VA and BA. These include median arteries and short and long lateral circumferential arteries which supply the brainstem tissue. All of the perforating branches converge to the aqueduct and the fourth ventricle.³ The pontine paramedian arteries supply the anteromedial aspects

of the pons.¹ Short circumferential branches from the BA supply the anterolateral aspects of the pons. The pontine long circumferential arteries arise not only from the basilar arteries but also from the AICA and the SCA.² The medial tegmental region has a prominently rich collateral supply, making it more resistant to ischemia than the base or lateral tegmentum.³

Penetrating branches from proximal PCA and SCA supply the paramedian midbrain and diencephalon. Much of the lateral aspects of midbrain is supplied by the SCA.²¹ The thalamus is supplied by perforating branches from the mesencephalic PCA and posterior communicating arteries.²¹ The thalamoperforating branches, issued from the posterior communicating and mesencephalic arteries and the thalamogeniculate arteries issued from the PCA supply the major part of the thalamus.^{2,22} The latter also supplies the posterior limb of the internal capsule. Occasionally, the right and left thalamoperforating arteries arise from a common single trunk that originates from the P1 segment of the PCA on one side (named the artery of Percheron.^{3,21,22} The lateral portions of the thalamus are supplied by a series of thalamogeniculate arteries.^{3,22} Occlusion of the thalamogeniculate branch of the PCA may result in thalamic syndrome because of infarction in the portion of thalamus that receives pain and temperature perception impulses.⁵ The medial and lateral posterior choroidal arteries (issuing from the mesencephalic arteries) end in the periventricular parts of thalamus.^{2,21} One of these branches supplies the anterior thalamic nucleus, the only nucleus of the thalamus supplied by a single vascular source.³ The tree-like fashion of distribution and their long course make the perforating branches particularly vulnerable to occlusive disease and can explain the high incidence of lacunes in basal ganglia and thalamus.³ Two important anatomic facts explain why VA origin lesions seldom cause chronic hemodynamically significant low flow to the vertebrobasilar system.¹⁷ First, the VAs are paired vessels that unite to form a single basilar artery. Second, the extracranial VA gives off numerous

muscular and other types of branches as it ascends in the neck.^{4,17} In contrast, there are no nuchal branches of the ICA. Thus, in the VA system, there is much more potential for development of adequate collateral circulation. Even when there is bilateral occlusion of the VAs at their origins, patients do not often develop posterior circulation infarcts.^{4,17,23} This interesting point could make influence in neurointerventional therapies because asymptomatic VA origin diseases are hemodynamically more benign than asymptomatic ICA origin disease.⁵ Atherosclerotic stenosis is more frequent in VA origin and distal segments of intracranial VAs and extends to the proximal portion of the BA.²⁴ Sever atherosclerotic stenosis is rare in cervical portions of VAs except on their origin.²⁵ In patients with VA origin stenosis, a bruit can often be heard over the supraclavicular region.¹⁷ Sometimes, a bruit is heard over the contralateral side because of increased collateral blood flow.¹⁷ The PCA arises as continuations of the mesencephalic arteries, passes above the oculomotor nerves, and moves posteriorly in a curvilinear manner laterally around the midbrain.¹ P1 segment of the PCA has an average diameter of 2.1 mm (0.7-3.0 mm) and runs anterolaterally for 5-10 mm to the PCoA. Then, the subsequent P2 segment of the PCA (average diameter: 2.3 mm; range: 1.3-3.0 mm) winds laterally and posteriorly around the cerebral peduncle.⁴ Soon after their origin, they anastomose with the PCoA to complete the circle of Willis. The PCoA runs anteriorly and laterally to connect the PCA to the ICA. The cortical branches of PCA nourish inferior and medial aspects of the temporal and occipital lobes to terminate at the visual cortex and occipital pole.¹ In 22% of people, the PCoA is hypoplastic and may even be aplastic on one side in 1%.^{5,25} Rare cases may be present with bilateral aplasia of the PCoA. The caliber of PCoA is inversely proportional to that of the P1 segment of the ipsilateral PCA. The average length of the PCoA is 14 mm (8-18 mm) and its average diameter is 1.2 mm (0.5-3.25 mm).⁴ Discussion about numerous neurovascular syndromes of vertebrobasilar territory is out of scope of this review article.

References

- Toole JF, Murros K, Veltkamp R. Cerebrovascular Disorders. 5th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 1999.
- Gillilan LA. Anatomy and embryology of the arterial system of the brain stem and cerebellum. In: Vinken PJ, Bruyn GW, editors. Handbook of clinical neurology. Amsterdam, The Netherlands: 1975. p. 24-44.
- Amarenco P, Caplan LR, Pessin MS. Vertebrobasilar occlusive disease. In: Barnett HJM, Mohr JP, Stein BM, et al, editors. Stroke: pathophysiology, diagnosis and Management. 3rd ed. New York, NY: Churchill Livingstone; 1998. p. 519-97.
- Baumgartner RW. Handbook on Neurovascular Ultrasound. Frontiers of Neurology and Neuroscience Series. New York, NY: Karger; 2006.
- DeReuck J. Embryologic and anatomic bases of human brain/spinal cord circulation and stroke. In: Ginsberg MD, Bogousslavsky J, editors. Cerebrovascular Disease: Pathophysiology, Diagnosis and Management. Malden, MA: Wiley-Blackwell; 1998. p. 935-8.
- Duvernoy HM. Human Brainstem Vessels. Berlin, Germany: Springer-Verlag; 1978.
- Gillilan LA. The correlation of the blood supply to the human brain stem with clinical brain stem lesions. J Neuropathol Exp Neurol. 1964; 23: 78-108.
- Amarenco P, Haww JJ. Cerebellar infarction in the territory of the anterior and inferior cerebellar artery. A clinicopathological study of 20 cases. Brain. 1990; 113(Pt 1): 139-55.

9. Amarenco P, Roullet E, Hommel M, et al. Infarction in the territory of the medial branch of the posterior inferior cerebellar artery. *J Neurol Neurosurg Psychiatry*. 1990; 53(9): 731-5.
10. Barth A, Bogousslavsky J, Regli F. Infarcts in the territory of the lateral branch of the posterior inferior cerebellar artery. *J Neurol Neurosurg Psychiatry*. 1994; 57(9): 1073-6.
11. Adams rd. Occlusion of the anterior inferior cerebellar artery. *Arch NeurPsych*. 1943; 49(5): 765-70.
12. Amarenco P, Hauw JJ, Gautier JC. Arterial pathology in cerebellar infarction. *Stroke*. 1990; 21(9): 1299-305.
13. Amarenco P, Roullet E, Goujon C, et al. Infarction in the anterior rostral cerebellum (the territory of the lateral branch of the superior cerebellar artery). *Neurology*. 1991; 41(2 (Pt 1)): 253-8.
14. Chaves CJ, Caplan LR, Chung CS, et al. Cerebellar infarcts in the New England Medical Center Posterior Circulation Stroke Registry. *Neurology*. 1994; 44(8): 1385-90.
15. Caplan LR. Occlusion of the vertebral or basilar artery. Follow up analysis of some patients with benign outcome. *Stroke*. 1979; 10(3): 277-82.
16. Caplan LR. *Posterior Circulation Disease*. Oxford, UK: Blackwell Science Inc.; 1996.
17. Caplan LR. *Caplan's Stroke: A Clinical Approach*. 3rd ed. Boston, MA: Butterworth-Heinemann Limited; 2000.
18. Ackerstaff RG, Eikelboom BC, Moll FL. Investigation of the vertebral artery in cerebral atherosclerosis. *Eur J Vasc Surg*. 1991; 5(3): 229-35.
19. Davison CH, Goodhart P, Savitsky N. The syndrome of the superior cerebellar artery and its branches. *Arch NeurPsych*. 1935; 33(6): 1143-74.
20. Bruckmann H, Ferbert A, del Zoppo GJ, et al. Acute vertebral-basilar thrombosis. Angiologic-clinical comparison and therapeutic implications. *Acta Radiol Suppl*. 1986; 369: 38-42.
21. Caplan LR, DeWitt LD, Pessin MS, et al. Lateral thalamic infarcts. *Arch Neurol*. 1988; 45(9): 959-64.
22. Bogousslavsky J, Caplan LR. *Vertebrobasilar Occlusive Disease: Review of Selected Aspects*. *Cerebrovasc Dis*. 1993; 3: 193-205.
23. Fields WS. Collateral circulation in cerebrovascular disease. In: Vinken P, Bruyn G, editors. *Handbook of clinical neurology*. Amsterdam, The Netherlands: North-Holland; 1972. p. 168.
24. Fisher CM. Occlusion of the vertebral arteries. Causing transient basilar symptoms. *Arch Neurol*. 1970; 22(1): 13-9.
25. Caplan LR, Wolpert SM. Angiography in patients with occlusive cerebrovascular disease: views of a stroke neurologist and neuroradiologist. *AJNR Am J Neuroradiol*. 1991; 12(4): 593-601.