Posteroinferior Cerebellar Artery Aneurysms: Influence of Angioanatomy on the Safety of Flow Diversion Treatment

Abstract

Several anatomical variables critically influence therapeutic strategies for posteroinferior cerebellar artery (PICA) aneurysms and, specifically, the safety of flow diversion for these lesions. We review the microsurgical anatomy of the PICA, discussing and detailing these considerations in the treatment of aneurysms of this vessel from a theoretical perspective and in light of our previously published clinical results.

Keywords: *Anatomy, endovascular, flow diversion, pipeline, posteroinferior cerebellar artery*

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Introduction

The vertebrobasilar circulation is a complex network of vessels bearing significant organizational homology to, and interconnections with, the spinal arterial system. The posteroinferior cerebellar artery (PICA) is a critical branch arising from the vertebral artery (VA); supplying the dorsolateral medulla and cerebellar tonsils, vermis, and hemispheres; and forming extensive anastomoses with other locoregional vessels.[1] An understanding of neurovasculogenesis illumines normal and variant PICA origins; segmental topology; regional vascular microsurgical anatomy; and perforator, anastomosis, and collateral supply.[1-3] Optimization of PICA aneurysm treatment is thus facilitated by an intimate appreciation for these relationships and precise characterization of lesional angioarchitecture.[4]

PICA is divided into anterior medullary, lateral medullary, tonsillomedullary, telovelotonsilar, and cortical segments[1] [Figures 1 and 2]. It provides cortical branches to the cerebellar hemispheres, vermis, and tonsils; supplies the choroid plexus of the 4th ventricle, along with the anteroinferior cerebellar artery (AICA) and superior cerebellar artery (SCA); and, importantly, provides critical perforators, which penetrate the medulla directly following takeoff from PICA or course over its surface for short (short circumflex perforators) or long (long circumflex perforators) distances, supplying highly eloquent parenchyma [Figure 3].

Several factors render microsurgical clipping[5] or reconstruction with bypass as preferred treatment modalities for many PICA aneurysms. These lesions often present ruptured with subarachnoid hemorrhage and are frequently friable or fusiform, requiring flow replacement via bypass. Furthermore, PICA occlusion or intraprocedural rupture during coil embolization may result in catastrophic deficits.[6] However, endovascular therapy has been used effectively in the treatment of these lesions,[4,7,8] and new interventional modalities, such as flow diversion (FD) (Chow *et al.*, 2012),[4,9,10] eschew many of the shortcomings of traditional endovascular approaches,[6,11,12] with significantly better safety (lower complication rates) comparable to superior efficacy (higher obliteration rates). In this review, we discuss the microsurgical anatomy of PICA and implications on considerations for treating aneurysms of this vessel via flow-diverting stents.

Variant Origins

PICA most frequently originates from the intradural VA in the majority of individuals above and, in some instances, below the level of the foramen magnum. Occasionally, PICA originates from the basilar artery or a segmental radiculopial artery at C1 or

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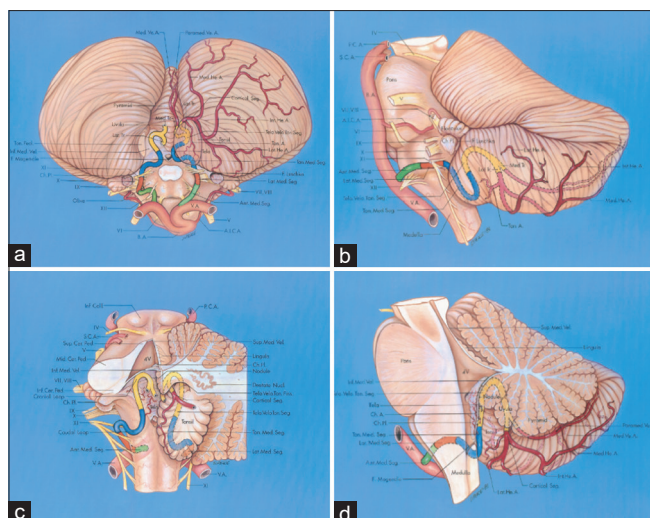*E-mail: mgg26@drexel.edu*

Figure 1: Posteroinferior cerebellar artery segments and course. (a) inferior view. The left tonsil has been removed at the level of the tonsillar peduncle. The posteroinferior cerebellar artery originates from the posterior, medial, or posteromedial surface of the vertebral artery. Its anterior (green) and lateral (orange) medullary segments course in the cerebellomedullary cistern posteriorly toward the inferior olive and lower cranial nerves. The tonsillomedullary segment (blue) of the posteroinferior cerebellar artery, originating just distal to the emergence of lower cranial nerves from the postolivary sulcus, courses toward the inferior pole of the tonsil, forming the caudal loop, then superiorly, as it transitions into the telovelotonsillar segment (yellow), forming the rostral loop, before becoming the cortical segment (red) and coursing distally to supply branches to the posteroinferior surface of the cerebellar hemispheres. Anterior medullary segment (green); lateral medullary segment (orange); tonsillomedullary segment (blue); telovelotonsillar segment (yellow); cortical segment (red). (b) enlarged posterior view. The left and part of the right halves of the cerebellum have been removed. (c) lateral view. (d) midsagittal section. A – Artery; AICA – Anteroinferior cerebellar artery; Ant – Anterior; BA – Basilar artery; Cer – Cerebellar; Ch – Choroid; Coll – Colliculus; F – Foramen; Fiss – Fissure; He – Hemispheric; Inf – Inferior; Int – Intermediate; Lat – Lateral; Med – Medial, medullary; Mid – Middle; Nucl – Nucleus; Paramed – Paramedian; PCA – Posterior cerebral artery; Ped – Peduncle; Pl – Plexus; SCA – Superior cerebellar artery; Seg – Segment; Sup – Superior; Ton – Tonsillar; Tr – Trunk; VA – Vertebral artery; Ve – Vermian; Vel – Velum. Modified with permission from Figure 2.15 of Rhoton, 2000[3]
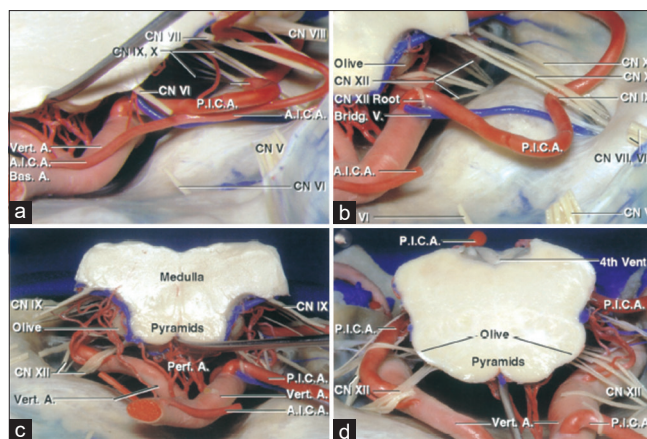

Figure 2: Cerebellar arteries. (a) Superolateral view. The posteroinferior cerebellar artery originates from the posterior surface of the vertebral artery, coursing in the cerebellomedullary cistern dorsally toward the olive and cranial nerves, exiting the postolivary sulcus, below glossopharyngeal and vagus nerves and above accessory nerve. (b) Enlarged superolateral view. The anteroinferior cerebellar artery and the nerves entering the internal acoustic meatus have been divided to better demonstrate posteroinferior cerebellar artery course and relationship to the lower cranial nerves. (c) Superior view: section at the level of the cochlear nuclei and inferior cerebellar peduncle of the medulla. Medullary perforators originating from the posteroinferior cerebellar, vertebral, and basilar arteries are well demonstrated. (d) Medullary section at the level of the hypoglossal nuclei. Modified with permission from Figure 2.5 of Rhoton, 2000[3]

C2, with the latter variants irrigating principally the tonsils, vermis, and posteroinferior surface of the cerebellum, with dorsolateral medullary supply being provided by PICA perforators and a prominent medullary perforator emanating from the VA in the stead of PICA.[1,2] The lateral spinal artery may originate from the VA as well as variant-origin PICAs.[2,13] Compromise of vascular territory supplied by variant C1 or C2 origin PICA, when treating aneurysms of this vessel, produces a different spectrum of clinical findings compared to occlusion of a VA-originating PICA, manifesting principally with cerebellar neurological signs and symptoms and typically lacking medullary deficits. Another common variant is the common origin, i.e., AICA–PICA complex, and occasionally bihemispheric PICA.[14]

It is important to recognize these embryologic variants and the vascular territory supplied when weighing the benefits against the potential risks of different therapeutic modalities in the treatment of PICA aneurysms. Coil embolization of an aneurysm resulting in occlusion of a variant origin or bihemispheric PICA may result in more severe and/or less characteristic deficits. Occlusion of an AICA–PICA complex may result in ischemia sparing the medulla, as this variant is associated with absence of bulbar perforator supply.[2] Occlusion of PICA originating extradurally at the C1 level may result in ischemia to the posterior, but not lateral, medullary surface. An increased risk of catastrophic stroke with iatrogenic PICA coil embolization of a bihemispheric PICA may shift the therapeutic decision toward selecting reconstruction with bypass or microsurgical clipping, permitting definitive obliteration and the ability to intraoperatively assess and ensure preserved PICA patency. FD-related PICA origin jailing, variably resulting in occlusion, is typically well tolerated for normal-origin PICA, with none of such treated patients suffering stroke,[4] which may be less well tolerated in the case of variant-origin PICAs.

Collateral Network

In the case of proximal ipsilateral VA occlusion, PICA may receive flow from the contralateral VA, anterior circulation via the basilar artery, anterior and lateral spinal arteries (arising from VA or PICA and anastomosing with other spinal vessels), and posterior meningeal artery.[2,15,16] Should occlusion of the PICA occur, flow is provided by rich collaterals among all cerebellar arteries, including the AICA and SCA and contralateral PICA (PICA–PICA) collaterals,[2] evidenced by the absence of ischemic deficit in patients with PICA aneurysms treated with FD stents
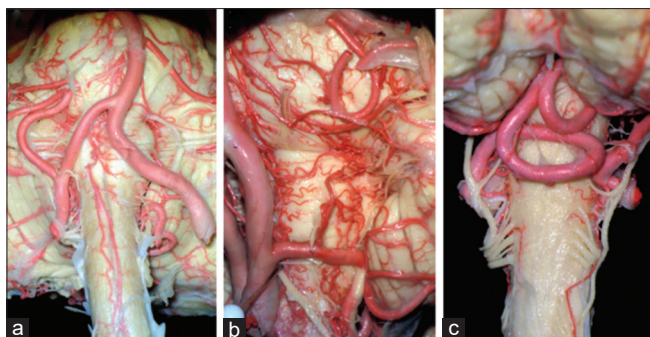
Figure 3: (a) Anterior view depicting three posteroinferior cerebellar artery origin variants. The classic origin from the vertebral artery is depicted on the right, and origins from the anteroinferior cerebellar artery–posteroinferior cerebellar artery complex from the basilar artery and extradural origin of posteroinferior cerebellar artery at C1 are depicted on the left. (b) Pial anastomosis on the olivary surface between branches of posteroinferior cerebellar artery and superior cerebellar artery. (c) Posteroinferior cerebellar arteries originating at C1 bilaterally, both of which give rise to the lateral spinal arteries. Modified with permission from Figures 1-9 of Mercier *et al.*, 2008[2]

jailing the PICA.[4,9] In a microscopic study, extensive anastomoses were observed over the inferior olivary surface in approximately half of the cases, with PICA shown to anastomose with SCA, AICA, and basilar branches.[2] This collateral supply would be most critical in providing supply to the perforator-irrigated PICA territory in cases of occlusion at the origin or distally.

In a series of PICA aneurysms, PICA remained patent in all cases wherein delayed postinterventional angiography was performed following FD treatment.[9] As a double-edged sword, these collaterals may also prevent aneurysmal obliteration or cause recurrence of a previously resolved lesion.[8,11,12] In a series of ten patients with PICA aneurysms treated with FD stents placed in VA or PICA, complete obliteration and partial reduction occurred in eight and two patients, respectively.[4] Collateral flow may have accounted for the two instances of incomplete obliteration.

Perforator System

The dorsolateral medulla is extensively irrigated by a rich anastomotic perforator network supplied from both the VA and the initial segments of PICA[1] [Figure 3]. According to one microdissection study, the anterior-segment perforators ranged from 0 to 2 in number and emanated from the superior, posterior, and medial surfaces of PICA; lateral-segment perforators ranged from 0 to 5 in number and arose from the medial surface of PICA; and tonsillomedullary perforators were the most numerous, ranging from 0 to 11 in number and emanated from the anterior and medial surfaces of PICA. Perforators are also supplied by the AICA and descending branches from the SCA. The VA medullary perforators were found to arise both proximally and distally with respect to the PICA origin,[1] with the latter noted more commonly. Consistently, VA perforators proximal to PICA origin were for the most part found to be absent according to a study by Lasjaunias

et al.[2,13] This group also demonstrated that the extent of brainstem perforator supply from the VA was in equilibrium with that provided by PICA: the more proximal the PICA origin, the more extensive the medullary perforator supply which is provided by VA. Thus, risk to perforator territory with FD jailing of PICA is theoretically diminished with more proximal origin of the PICA from the VA. In practice, however, PICA patency is maintained despite jailing of its origin.[9,17] Furthermore, chronic FD jailing-related occlusion is better tolerated than acute occlusion.[18] In three cases of PICA aneurysms treated with FD stents placed wholly within PICA, no patients suffered stroke.[4]

Compromise of PICA or VA perforators could precipitate ischemia and/or infarction of critical medullary regions subserving a myriad of functions,[1,19-22] manifesting clinically as Wallenberg syndrome. The majority of PICA aneurysms occur at the origin and occlusion here would critically compromise the medullary perforators. While retrograde collateral supply of PICA, from the lateral spinal artery, for instance, may in theory fill the PICA medullary perforators in the case of a PICA origin occlusion, FD would compromise perforators throughout the stents' expanse/lay. Any compensation for perforator occlusion/compromise would have to be via (1) perforator redundancy from other vessels supplying the same region and/or (2) perforator anastomoses; the extent to which either can compensate effectively for PICA perforator occlusion remains to be elucidated. More porous nonflow-diverting stents would be convenient in this regard, but PICA tortuosity is generally prohibitive of their use in this location. Treatment of distal PICA aneurysms puts at risk the parenchyma of comparatively lower "eloquence-density," with respect to perforator compromise via flow-diverting stents or main vessel stenosis or occlusion via in-stent thrombosis or coil migration, etc.

In a previous series, we classified PICA aneurysms according to (1) morphology and (2) location.[4] Saccular and fusiform aneurysms were designated as Types 1 and 2, respectively. Saccular aneurysms involving the PICA–VA junction or those on the proximal 5 mm of PICA were designated as Type 1a and 2a and those located distally as Type 1b and 2b. Fusiform PICA aneurysms arising distal to the medullary perforator supply were designated as Type 2c. Type 1a and 2a lesions are treated by placement of a flow diverter in the VA, whereas Type 1b and 2b aneurysms are amenable to treatment by placement of the entire flow-diverting stent in PICA.[4] In proximal VA-origin PICA, with more extensive perforator supply from the VA, FD placement within PICA is theoretically associated with less risk of perforator territory ischemia. Thus, the ideal Type 1b and 2b lesions would involve a proximally originating PICA. Conversely, when treating a Type 1a or 2a PICA aneurysm, requiring placement of the FD stent within the VA, PICA originating from more distal aspects of the VA, associated with greater perforator supply

deriving from PICA, is associated with less theoretical risk of perforator territory ischemia; in this instance, should PICA undergo a "tourniquet occlusion" at its origin, the nonjailed PICA perforators would fill retrogradely from anastomoses (e.g., ipsilateral AICA and contralateral PICA).

Three patients in our series of PICA aneurysms in whom a flow-diverting stent was placed wholly within PICA suffered no medullary infarction.[4] Moreover, in patients with VA dissecting aneurysms, no instances of perforator territory ischemia occurred with neuroform or coronary stenting[23] or FD.[24] In contrast, in one series, treatment with internal coil trapping resulted in medullary infarction in 50% of patients with VA dissection, including 60% of patients in whom occipital artery–PICA revascularization was performed, even though the anastomosis was well placed on the caudal loop of the tonsillomedullary segment of the PICA to supply the medullary perforators.[19] Thus, FD and stenting proved better at protecting the VA–PICA perforator network compared to revascularization.

Conclusion

The heterogeneity of PICA anatomy renders preoperative characterization of angioarchitecture and locoregional anatomy critical in the treatment of PICA aneurysms. PICA aneurysm classification according to morphology and location allows appropriate selection of location for FD placement to obliterate the aneurysm. The presence of collateral supply renders PICA origin and/or perforator ostium FD-related jailing classically well-tolerated whether or not chronic occlusion occurs. This is validated by patients of a previous series suffering no instances of stroke with placement of FD in VA or PICA.[4,9] However, an appreciation for exceptions to this rule and instances in which treatment via FD may prove too risky is critical and is predicated upon an ability to predict the extent to which collateral supply will be able to effectively compensate for jailing of PICA origin (i.e., with FD in VA) or perforator ostia (i.e., with FD in PICA). Large-scale studies are necessary to validate the practicality of the aforementioned theoretical anatomical considerations in the treatment of PICA aneurysms and the safety and efficacy of FD for the same.

Conflicts of interest

There are no conflicts of interest.

References

1. Lister JR, Rhoton AL Jr., Matsushima T, Peace DA. Microsurgical anatomy of the posterior inferior cerebellar artery. Neurosurgery 1982;10:170-99.
2. Mercier PH, Brassier G, Fournier HD, Picquet J, Papon X, Lasjaunias P. Vascular microanatomy of the pontomedullary junction, posterior inferior cerebellar arteries, and the lateral spinal arteries. Interv Neuroradiol 2008;14:49-58.
3. Rhoton AL Jr. The cerebellar arteries. Neurosurgery 2000;47:S29-68.
4. Srinivasan VM, Ghali MG, Reznik OE, Cherian J, Mokin M, Dumont TM, *et al*. Flow diversion for the treatment of posterior inferior cerebellar artery aneurysms: A novel classification and strategies. J Neurointerv Surg 2018;10:663-8.
5. Lawton MT. Seven Aneurysms Tenets and Techniques for Clipping. 1st ed. New York: Thieme; 2011. p. xiii, 224.
6. Xu F, Hong Y, Zheng Y, Xu Q, Leng B. Endovascular treatment of posterior inferior cerebellar artery aneurysms: A 7-year single-center experience. J Neurointerv Surg 2017;9:45-51.
7. Juszkat R, Kram P, Stanisławska K, Jankowski R, Stachowska-Tomczak B, Nowak S, *et al*. Ten years of experience in endovascular treatment of ruptured aneurysms of the posterior inferior cerebellar artery. Interv Neuroradiol 2016;22:129-37.
8. Mascitelli JR, Yaeger K, Wei D, Kellner CP, Oxley TJ, De Leacy RA, *et al*. Multimodality treatment of posterior inferior cerebellar artery aneurysms. World Neurosurg 2017;106:493-503.
9. Levitt MR, Park MS, Albuquerque FC, Moon K, Kalani MY, McDougall CG. Posterior inferior cerebellar artery patency after flow-diverting stent treatment. AJNR Am J Neuroradiol 2016;37:487-9.
10. Chow M, McDougall C, O'Kelly C, Ashforth R, Johnson E, Fiorella D. Delayed spontaneous rupture of a posterior inferior cerebellar artery aneurysm following treatment with flow diversion: A clinicopathologic study. AJNR Am J Neuroradiol 2012;33:E46-51.
11. Crowley RW, Albuquerque FC, Ducruet AF, Williamson RW, McDougall CG. Technical considerations in the endovascular management of aneurysms of the posterior inferior cerebellar artery. Neurosurgery 2012;71:ons204-17.
12. Chalouhi N, Jabbour P, Starke RM, Tjoumakaris SI, Gonzalez LF, Witte S, *et al*. Endovascular treatment of proximal and distal posterior inferior cerebellar artery aneurysms. J Neurosurg 2013;118:991-9.
13. Lasjaunias P, Vallee B, Person H, Ter Brugge K, Chiu M. The lateral spinal artery of the upper cervical spinal cord. Anatomy, normal variations, and angiographic aspects. J Neurosurg 1985;63:235-41.
14. Cullen SP, Ozanne A, Alvarez H, Lasjaunias P. The bihemispheric posterior inferior cerebellar artery. Neuroradiology 2005;47:809-12.
15. Arai A, Miyamoto H, Ashida N, Kohmura E. Spontaneous occlusion of PICA-involved dissecting aneurysm with development of a collateral channel from the posterior meningeal artery. No Shinkei Geka 2012;40:997-1002.
16. Chang SW, Kakarla UK, Sharma GK, Spetzler RF. Posterior inferior cerebellar artery origin thrombosis with aneurysm of collateralized posterior meningeal artery presenting as subarachnoid hemorrhage: Case report. Neurosurgery 2009;65:E818-9.
17. Mazur MD, Kilburg C, Wang V, Taussky P. Pipeline embolization device for the treatment of vertebral artery aneurysms: The fate of covered branch vessels. J Neurointerv Surg 2016;8:1041-7.
18. Srinivasan VM, Mokin M, Duckworth EAM, Chen S, Puri A, Kan P. Tourniquet parent artery occlusion after flow diversion. J Neurointerv Surg 2018;10:122-6.
19. Endo H, Matsumoto Y, Kondo R, Sato K, Fujimura M, Inoue T, *et al*. Medullary infarction as a poor prognostic factor after internal coil trapping of a ruptured vertebral artery dissection.

J Neurosurg 2013;118:131-9.

20. Hamada J, Kai Y, Morioka M, Yano S, Todaka T, Ushio Y. Multimodal treatment of ruptured dissecting aneurysms of the vertebral artery during the acute stage. J Neurosurg 2003;99:960-6.

21. Iihara K, Sakai N, Murao K, Sakai H, Higashi T, Kogure S, *et al.* Dissecting aneurysms of the vertebral artery: A management strategy. J Neurosurg 2002;97:259-67.

22. Lee JM, Kim TS, Joo SP, Yoon W, Choi HY. Endovascular treatment of ruptured dissecting vertebral artery aneurysms – Long-term follow-up results, benefits of early embolization, and predictors of outcome. Acta Neurochir (Wien) 2010;152:1455-65.

23. Park SI, Kim BM, Kim DI, Shin YS, Suh SH, Chung EC, *et al.* Clinical and angiographic follow-up of stent-only therapy for acute intracranial vertebrobasilar dissecting aneurysms. AJNR Am J Neuroradiol 2009;30:1351-6.

24. Narata AP, Yilmaz H, Schaller K, Lovblad KO, Pereira VM. Flow-diverting stent for ruptured intracranial dissecting aneurysm of vertebral artery. Neurosurgery 2012;70:982-8.