ORIGINAL RESEARCH

The bihemispheric posterior interior cerebellar artery: anatomic variations and clinical relevance in 11 cases

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ABSTRACT

Purpose Although the anatomic course of the posterior interior cerebellar artery (PICA) is variable, it is thought to be very rare for the artery to cross midline, with an estimated incidence of 0.1%. Bihemispheric PICA crosses midline and typically serves both PICA territories.

Methods We present 11 cases of bihemispheric PICA discovered from retrospective angiogram review, the largest to date reported in the literature.

Results Five cases were the typical bihemispheric PICA pattern, three were bihemispheric with distal vertebral hypoplasia, two cases were the vermician type, and one was atypical, with the PICA feeding a contralateral cerebellar arteriovenous malformation (AVM). The branching point to the contralateral hemisphere always occurred distal to the ascending tonsillar loop and all true bihemispheric variants had contralateral PICA aplasia.

Conclusions The true incidence of this variant may be much higher than previously thought (3.6% in the current series), and has relevance for cerebrovascular disease, including aneurysm, AVM, and ischemic stroke. Neuroradiologists, neurologists, and neurosurgeons should be aware of this potential variant.

BACKGROUND

Numerous variations of the posterior interior cerebellar artery (PICA) have been described but only a few cases of a single PICA serving bilateral cerebellar hemispheres have been reported. Cullen et al have estimated the incidence at less than 0.1%. When one PICA is hypoplastic or aplastic, the PICA territory is typically supplied by an enlarged anterior inferior cerebellar artery (AICA) or superior cerebellar artery (SCA). The so-called bihemispheric PICA supplies both cerebellar hemispheres, and has clinical relevance, especially in cases of PICA aneurysm, cerebellar arteriovenous malformation (AVM), and bilateral cerebellar infarction. This variant is further subcategorized into the true bihemispheric PICA, serving the contralateral PICA territory, and the vermician variant, serving only the midline. In the true bihemispheric variant, there should be aplasia of the contralateral PICA, although in the vermician variant, the contralateral PICA may be present. It has been hypothesized that the true incidence of the finding may be higher than reported but the distal location of the crossing may make it easy to confuse a bihemispheric PICA with normal distal filling in the cerebellum from the contralateral AICA, SCA, or PICA. We describe a series of 11 patients with bihemispheric PICA identified on cerebral angiography, and discuss the angiographic findings and clinical relevance.

CASE SERIES

Following institutional review board approval, we identified 11 cases of bihemispheric PICA. We initially observed the variant in two very clear cases. One of these two cases was clinically relevant in that the contralateral PICA was feeding an AVM in the cerebellar hemisphere. We then retrospectively reviewed 250 consecutive angiograms specifically evaluating for the presence of the bihemispheric PICA, and nine additional cases were identified for an estimated incidence of 3.6% (9/250). Only angiograms with bilateral adequate PICA visualization with multiple oblique projections were considered. Except for the AVM patient, all cases were incidental.

All of the true bihemispheric variants (n=8) supplied portions of the contralateral PICA territory and were associated with aplasia of the contralateral PICA. The two vermician variants served only the vermis and contralateral medial cerebellar hemisphere and both cases had contralateral PICAs present. All of the branching points to the contralateral hemisphere were distal along the course of the artery, typically after the second ascending loop of the vessel in the region of the superior pole of the tonsil distal to the choroidal point. The final case is similar in its bihemispheric nature, but is an atypical example with dilatation of a contralateral PICA feeding vessel to an AVM. Consistent with the previous cases, the crossing point is distal along the course of the vessel.

Case Nos 1–5: true bihemispheric PICA
All of these cases were incidentally discovered (figure 1A–H). In each case, there was aplasia of the contralateral PICA with no capillary filling of the cerebellar hemisphere visualized from the vertebral artery contralateral to the bihemispheric PICA. The anomaly was best visualized in the very early arterial phase (before there was significant basilar filling as with case No 2, figure 1C). The branching point in every case was distal to the tonsillar loop and ascents to the medullary vein, often after the vessel turned again around the rostral portion of the tonsil. Oblique projections were essential to show the branching points as well as the capillary filling of both cerebellar hemispheres from the single PICA.
Case Nos 6–8: bihemispheric PICA with distal vertebral aplasia or hypoplasia

These cases represent the same variant as above but are more readily seen angiographically due to hypoplasia or aplasia of the distal cranial vertebral artery (figure 2A–C). All cases were associated with the same key features as described above, with contralateral PICA aplasia and a distal crossing point after the tonsillar loop. In case No 6, there was near aplasia of the distal vertebral artery and most of the cerebellum filled from the left PICA (figure 2A), essentially as a continuation of the vertebral artery. In case No 7 (figure 2B), the right vertebral artery served both PICA territories and filled the basilar apex via SCA collaterals. The contralateral vertebral artery was occluded distally prior to the vertebrobasilar junction. In case No 8, the bihemispheric PICA arose from a hypoplastic vertebral artery. This again made the relatively small crossing vessel of the distal PICA well visualized (figure 2C).

Case Nos 9 and 10: vermian variant of bihemispheric PICA

In these cases, only the medial structures along the vermis and medial surface of the contralateral cerebellum were supplied by the vessel (figure 3A, B). In case No 9 (figure 3A), paired vessels were seen coursing in the midline to the distal cerebellar cortex along the vermis from the PICA. The ipsilateral PICA territory filled normally but no filling was seen in the region of the contralateral PICA. On the contralateral injection, there was a normal PICA filling the expected territory. The crossing vessel appeared to only anastomose with the distal SCA branches from the contralateral side. In case No 10 (figure 3B), the finding is very subtle and so we would classify this as a ‘probable’ vermian variant of the bihemispheric PICA. There is arterial filling of the right distal medial cerebellum from the left vertebral injection. The contralateral PICA is also present in this case.
Case No 11: atypical bihemispheric PICA

In this case, a left cerebellar AVM was evaluated for preoperative embolization (figure 4A–C). The left PICA was aplastic and the AVM was feeding primarily from a large left AICA/PICA, which was embolized. Control angiography showed continued filling, and although there appeared to be a vessel crossing midline, it was thought that this was superimposition or other angiographic cause. Eventually, selective catheterization of the right PICA was performed (figure 4A) and, surprisingly, found to be filling the AVM (figure 4C). This pedicle was then also embolized across midline. It is not clear whether this would have represented a bihemispheric or vermian variant, although as the AVM was in the cerebellar hemisphere in the PICA territory, the vessel likely embryologically would have been the true bihemispheric type. In addition, the contralateral PICA aplasia is consistent with our remaining series of bihemispheric types.

DISCUSSION

The typical course of PICA is one of the most variable intracranial arteries due to it being one of the last vessels to form phylogenetically and embryologically. The vessel is initially derived from a large radiculo-pial vessel serving the lateral medulla and only in late phylogenetic stages are distal vessels annexed for cortical supply to the cerebellum. Cullen et al liken the series of events to the recruitment of the anterior choroidal—PICA is initially recruited to supply the choroid of the fourth ventricle then the inferior cerebellum and vermis as these structures descend. For this reason, there is significant variation and collateralization between the PICA, AICA, and SCA.

Anatomically, the origin of PICA ranges from the distal cervical vertebral artery to the vertebrobasilar junction. The vessel initially usually ascends lateral to the olive in the cerebellomedullary cistern and is closely related to the lower cranial nerves. Small perforating vessels to the medulla may arise in this anterior and lateral medullary segment. The artery then descends to the tonsillar loop and then ascends to the posterior medullary velum (tonsillomedullary segment). A choroidal branch is nearly always present but is variable in terms of origin and morphology. This branch defines the choroidal point. The second superior loop is at the superior pole of the tonsil (the telovelontonsillar segment). After this point, the artery divides into variably named branches, including ‘medial and lateral’ or ‘tonsilohemispheric and vermian’, respectively. It is at this point that the bifurcation crossing midline occurred in all of our cases.

There are a number of different mechanisms by which intradural vessels can cross midline, including a crossing network of small pial vessels, a midline structure such as the commissure (or the vermis in this case) influencing a vessel to cross or, finally, fusion of midline structures such as with the basilar artery. It is thought that the presence of the midline vermis probably influences the midline communication of the PICA to supply the contralateral hemisphere. This also explains why the crossing point always occurs distally along the vessel in the vermian segment.

We present, to our knowledge, the largest series of angiographically proven bihemispheric PICA in the current literature. This better defines the incidence as around 3% and that the true bihemispheric type as four times as common as the vermian variant. With a true bihemispheric PICA, there is contralateral PICA aplasia whereas there is a normal PICA with the vermian variant. In the previous literature, the largest series was a series of four incidental cases reported by Cullen. One case of

Figure 3 Vermian variant of bihemispheric posterior interior cerebellar artery (PICA). (A) Magnified anteroposterior view of left vertebral injection in case No 9 showing the contralateral vermian branches (arrow) supplied by the left PICA. (B) AP projection of left vertebral injection in case No 10 showing the faint cross midline filling in the vermis.

Figure 4 Bilateral supply to a left cerebellar arteriovenous malformation (AVM) in case No 11. (A) Anteroposterior view of right vertebral injection. The AVM is seen on the left. The arrow shows the distal right posterior interior cerebellar artery (PICA). (B) Microcatheter injection from the right PICA. The arrow points to the same segment of PICA as in (A). AVM is filling across midline. (C) Intranidal microcatheter injection. The course of the microcatheter is traced in gray and can be compared with the initial angiogram (A) as the course of PICA.
aneurysm at the origin of a bihemispheric PICA has been reported. Several cases of ruptured aneurysms of the ‘PICA communicating artery’ have been reported. This terminology may not be accurate as the communication is not a normal anastomosis and in the reported cases there was typically aplasia of the contralateral PICA suggesting that there was in fact a bihemispheric PICA.

The clinical relevance for neurointerventionalists and neurosurgeons of this finding is significant. In cases of aneurysm of PICA, if the lesion is distal to the medullary segment, parent vessel sacrifice is often considered. If there is risk of bilateral cerebellar infarction, however, a different strategy such as bypass may have to be adopted, as this bilateral stroke would be more disabling than a unilateral distal PICA stroke. This may even apply to temporary clipping during aneurysm surgery. Based on our series, it seems that formal angiographic evaluation for bihemispheric PICA should be part of the preoperative workup for these lesions. The second clinically relevant case is with regard to AVMs. Although we present the only reported case in the literature, the possibility of bilateral supply to the cerebellar AVM is now raised and, again, specific attention should be paid to this finding as the surgical or embolization approach will be different if bilateral feeders could be present. Although a 3.6% incidence is low, it is significantly higher than the previously suggested <0.1%, and may therefore impact on vascular decision making.

Bihemispheric PICA may not have been well recognized historically due to limitations in resolution of angiography in the more distal vasculature. Furthermore, the typically very tortuous course of PICA and the relatively distal crossing points after the second ascending loop of PICA make the bihemispheric nature difficult to detect. Even on three-dimensional rotational angiography reconstruction, the detail of these smaller distal vessels may be lost. Although a ‘double vessel’ sign has been described on lateral angiography, we do not find this useful due to the tortuous and bilaterally variable course. A high index of suspicion needs to be held any time there is aplasia of one PICA. In these cases, the contralateral vertebral artery should be assessed in the early arterial phase for contralateral filling before filling of the contralateral AICA or PICA. Oblique projections can be very useful to see vessels filling both cerebellar hemispheres from one PICA. It would be unlikely to definitively demonstrate a bihemispheric PICA in non-invasive imaging due to the small caliber of the vessels and the inability to determine the dynamic filling pattern.

This higher incidence of bihemispheric PICA than previously reported is supported by data from the stroke literature noting a higher incidence of bilateral PICA stroke than previously recognized. Brusa et al identified a case of stroke in bilateral cerebellar hemispheres and hypothesized that the most likely explanation was a PICA serving both hemispheres. Gaida-Hommernick et al then reported a single case which established this link where bilateral cerebellar infarction was caused by stenosis of the origin of a proven bihemispheric PICA. Kang et al then challenged the concept that bilateral PICA infarct was rare by reporting a series of 12 patients with bilateral PICA territory infarction out of 40 consecutive patients with PICA territory stroke, suggesting that 30% of patients with PICA infarct may have some bilateral involvement. The authors report that 10 of the 12 patients had patterns that were most likely due to a bihemispheric PICA. This was based on the lack of cardiac source and the fact that in many of these there was unilateral vertebral disease on the side of the dominant PICA and the larger stroke, suggesting that this vessel was the source of the bilateral strokes rather than less likely explanations such as occlusion of the basilar artery, pressure effects, and simultaneous emboli. Our series establishes this radiologic confirmation—that the presence of bihemispheric PICA may be much higher than previously suspected.

CONCLUSION

Bihemispheric PICA is an unusual variant of the distal PICA. The true incidence of such anomaly may be 3.6%—much higher than previously thought and has clinical relevance for vascular disease, including AVM, aneurysm, and ischemic stroke. The branching point occurs after the tonsillar segment and so may be difficult to demonstrate angiographically. Neuroradiologists, neurologists, and neurosurgeons should be aware of this anatomical variant and potential clinical relevance.

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