Association of Two Primitive Carotid-Basilar Anastomoses and Cerebrovascular Abnormalities on the Brain Base: A Case Report

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ABSTRACT

Simultaneous presence of the persistent primitive trigeminal artery and so-called intermediate communicating artery was discovered in a 77-year-old cadaver autopsied due to the myocardial infarction. Many vascular variants and abnormalities such as aplasia of the right vertebral artery (VA), presence of two right posterior cerebral arteries (PCAs), partial duplication of the right superior cerebellar artery, hypoplasia of the pre-communicating part (A1) of the right anterior cerebral artery and the right PCA of basilar origin, a special configuration of the anterior communicating artery (ACoA), and a small aneurysm at the right A1–ACoA junction were associated. The finding of an incipient cerebral aneurysm at the junction of the hypoplastic A1 and embryonal configuration of the ACoA in the eighth decade of life indicates that its development was caused by long-term pressure of blood flow at branching points of this artery independent from its caliber. However, it is not yet clear whether the persistence of the first and/or the second carotid-basilar anastomoses in this case was the condition for an aplasia of one VA or vice versa.

KEYWORDS: Carotid-Basilar anastomosis, Cerebrovascular abnormality, Human brain

INTRODUCTION

During embryonic development of the cerebral vasculature, primitive carotid–vertebrobasilar anastomoses (CVBAs) — the trigeminal (PTA), otic (POA), hypoglossal (PHA), and preatlantal intersegmental (PIA) arteries, and caudal end of the internal carotid artery (ICA) bilaterally connect primitive ICAs and precursors of future basilar artery (BA) for a period of 7 to 10 days, when the human embryo reaches about 4-mm length (5,18).

A persistent PTA (PPTA), as cited (18), has been noted in 0.03% to 2.2% of cases. It was discovered in human fetuses (4), neonates and infants (9), in the fourth decade (3,22), or in the elderly—eighth (13,23) and ninth decade of life (19). The PPTA persisted singly and unilaterally (3,4,6,7,9,11,13,15,22,23), or bilaterally (1,8), or interacting with another CVBA (14,18,24). The PPTA and aplasia of some arteries such as the BA (6,11), or one vertebral artery (VA) (6,7,9,14,22), or both VAs (8,24), or one ICA (3,9), or other vascular variants (3,6,9,14,22), or visceral abnormalities (9) were associated. There may be aneurysms, as cited (18), in nearly 14% of all PPTA cases, as well as only 2% from PPTA itself.

An excess vessel between two posterior cerebral arteries on the brain base as “bridge” was sketched in the book of microneurosurgery (21), while the same vessel as “intermediate communicating artery” (ICoA) in human fetuses and adult cadavers was marked (16,17).

The purpose of this report was to present the PPTA associated with ICoA and other vascular abnormalities.
CASE REPORT

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. Experimental protocol and informed consent were approved by the Research Ethics Committee of the Faculty of Medicine in Niš (No. 01-206-1).

Two CVBAs and many vascular abnormalities on the brain base were found in a 77-year-old male cadaver autopsied due to the myocardial infarction.

The outer diameters (ODs) of corresponding cerebral arteries were studied with the digital images using the ImageJ program (http://rsb.info.nih.gov/ij/index.html).

The ODs of the left ICA (OD=3.97 mm) and right ICA (OD=3.87 mm) were similar to those of the left VA and BA (OD=3.84 mm). The left posterior communicating artery (PCoA) (OD=1.26 mm) and left PCA (OD=2.01 mm) were of normal size. There were also a network configuration of the anterior communicating artery (ACoA) and mild hypoplasia (OD = 1.37 mm) of the pre-communicating part (A1) of the right anterior cerebral artery (ACA); simultaneously, the left ACA (OD=3.41 mm) was bihemispheric in the post-communicating part. In addition, an incipient aneurysm at the right A1–ACoA junction and atheromatous plaques in the walls of both ICAs and left VA were associated pathological findings. We found supernumerary side branch (OD=1.97 mm) originating from the BA below the left superior cerebellar artery (SCA) and initially coursing parallel to the latter. The size of the BA caliber did not differ before and beyond this branch. The right SCA was partially duplicated at the beginning from the BA; the left posterior inferior cerebellar artery (PICA) began from the left VA, while the right PICA originated from the BA in this case (Figure 1 A,B).

As supernumerary BA side branch was artificially cut, and the left VA continued in the BA, the posterior and middle cranial fossae, as well as other associated vascular abnormalities on the brain base were investigated. Supernumerary BA branch actually originated from the cavernous part (C3) of the left ICA and penetrated the dura mater at the left edge of the dorsum sellae; it was the reason for marking this branch as a CVBA, i.e. the PPTA (Figure 2A). Simultaneously, the right VA was absent at the level of the foramen magnum.

Figure 1: View on some arteries of the carotid and vertebrobasilar systems on the brain base. A) Original picture shows configuration of main arteries on the ventral side of myelencephalon, pons, hypothalamus and frontal lobes of the telencephalon. Two carotid-vertebrobasilar anastomoses are pointed by arrows; an incipient aneurysm of the right A1–ACoA junction is marked by an asterisk. B) Main arteries and incipient aneurysm on the brain base are highlighted. (C4) cerebral part of the internal carotid artery; (A1) pre-communicating part of the anterior cerebral artery; (A2) post-communicating part of the anterior cerebral artery; (ACoA) anterior communicating artery; (RAH) Heubner’s recurrent artery; (M1) sphenoidal part of the middle cerebral artery; (PCoA) posterior communicating artery; (PCAc) posterior cerebral artery of carotid origin; (ICoA) intermediate communicating artery; (PCAb) posterior cerebral artery of basilar origin; (P1) pre-communicating part of the posterior cerebral artery; (P2) post-communicating part of the posterior cerebral artery; (SCA) Superior cerebellar artery; (PPTA) persistent primitive trigeminal artery; (ACoA) anterior inferior cerebellar artery; (BA) basilar artery; (PICA) posterior inferior cerebellar artery; (VA) vertebral artery.
Additionally, there was an extreme hypoplastic ICoA that connected two posterior cerebral arteries (PCAs) on the right side, i.e. PCA of carotid origin (OD=2.19 mm) and hypoplastic PCA (OD=0.75 mm) of basilar origin (Figure 2B).

**DISCUSSION**

The presented PPTA was the third case found during autopsy of adult cadavers in our population specimens; the first case was discovered during students’ exercises (15), while the second one was also discovered during forensic autopsy (18). The PPTA originated from the left C3 part, as in cases described by Yeniceri et al. (22), and Zargouni and Marichal (24).

Angiographic anatomy and classification of the PPTA in two types, as cited (18), was given by Saltzman almost sixty years before. In Saltzman type 1 PPTA, the BA proximal to the junction with the PPTA may be hypoplastic, and the bilateral PCoAs may be absent; the junction of Saltzman type 1 PPTA with the BA is below the origin of the SCA, and the PCA receives blood predominantly through the patent PCoAs, whereas the BA was completely filled by one or both VAs (1,18). The type 2 can have the junction above or below the SCA where the PPTA supplies the SCA and the contralateral PCA, while the PCoA supplies the ipsilateral PCA (5,18). The presented PPTA and status of above mentioned arteries indicate a new PPTA type. Namely, the size of BA caliber did not differ before and beyond the PPTA–BA junction, while the PCoA was absent unilaterally and replaced by an ICoA, and the BA was filled by one (left) VA.

We found that the left PPTA was associated with aplasia of the right VA as in a case described by Möller-Hartmann et al. (6). It is well known, as cited (18), that if the right and/or left VAs have not developed, the blood circulation of the posterior brain can be complementarily supplied by some of CVBAs, such as of persistent PTA in this and other cases (3,6,7,9,14,22), and/or the PIA (6,14), or the PHA (13), or the POA (10). The question is whether the persistence of the PTA in this case induced an aplasia of the VA, or vice versa, especially according to the fact that there were cases of unilateral aplasia of the VA and persistence of the PTA on opposite side (6,7,9). Additional question is a finding of simultaneous persistence of the PTA and PIA in a case of aplasia of ipsilateral VA (14), or a persistence of bilateral PTA in a case of aplasia of one VA (1).

If we take into account the case’s age, the question is why the PPTA was not pathologically changed, as it was in a 31-old female and a 59-year old male (12). However, the POA in a 70-year old-woman (10), or the PHA in a 79-year-old woman (13), or the PIA in a 71-year-old man (23), were not pathologically changed also.

In this, as well as in previous cases (16–18), the cerebral arterial circle (CAC) with an ICoA connecting two right PCAs can be compared with a decagon. We think that the right PCA of basilar origin is a continuation of primitive longitudinal neural artery on this side, while the PCA of carotid origin and ICoA were branches of a caudal end of primitive ICA. According to this opinion, we considered that ICoA is a CVBA. The presence of this supernumerary vessel in the CAC was proved in 7/200 fetuses (16), and 4/48 adult cadavers (17). The coexistence of the PPTA and ICoA in this and previous cases (18), or the PPTA and another CVBA (14,24), could be related to an underlying embryologic dysgenesis or a common insult resulting in vascular maldevelopment during the first several gestational weeks.

**Figure 2:** Two persistent carotid-vertebrobasilar anastomoses. A) Highlighted cavernous carotid (C3) origin of the left persistent primitive trigeminal artery (PPTA). PPTA perforation of the dura mater at the level of the left edge of the dorsum sellae, and the presence of single (left) vertebral artery (VA) at the level of the foramen magnum are shown. B) So-called intermediate communicating artery (ICoA) between the right posterior cerebral artery (PCAc) of carotid (C4) origin and the posterior cerebral artery (PCAb) of basilar (BA) origin is highlighted.
We found the hypoplastic right A1 part as associated vascular variant. As cited by Dimmick and Faulder (2), hypoplasia of an A1 part could be seen in 10% of autopsies, although we proved that hypoplasia of A1 part is rare in our population (19). Simultaneously, ACoA in this case was as a network, or using Latin language as “the rete communicans anterior” (20), and indicates the remnant of embryonal plexiform anastomosis of both ACAs.

Different vascular variants were associated with aplasia of one VA and persistence of some CVBA, such as hypoplasia of opposite VA (14), or BA (22), or ICA and PCA (9), and presence of variable branches of CVBA (9,14).

We also observed an incipient aneurysm at the right A1-ACoA junction in this case, while other authors described stenosis of the arch of the aorta (3), or ICA (6,7).

CONCLUSION

An association of the PPTA and ICoA and other vascular abnormalities, but without a difference in the BA caliber before and beyond BA-PPTA junction, could be related to a new type of PPTA, while ICoA should be included in the sheet of CVBAs. The finding of an incipient cerebral aneurysm at the junction of hypoplastic A1 and embryonal configuration of ACoA in the eighth decade of life indicates that its development was caused by long-term pressure of blood flow at branching points of this artery independently from its caliber. However, it is not yet clear whether the persistence of the first and/or the second CVBA in this case was the condition for the aplasia of one VA, or vice versa.

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