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Embryonic, anatomical, and clinical significance of persistent primitive intracranial arteries

Embriologiczne, anatomiczne i kliniczne znaczenie przetrwałych pierwotnych tętnic wewnątrzczaszkowych

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Abstract

Primitive intracranial arteries, including the trigeminal, proatlantal, otic, hypoglossal, and stapedial arteries, are embryonic vascular structures that temporarily connect the anterior and posterior, or intracranial and extracranial, circulations before typically regressing during development. Their persistence into adulthood, although rare, may alter local haemodynamics, contributing to aneurysm formation, vessel stenosis, or cranial nerve dysfunction. However, given the low incidence of associated pathologies, the mere presence of a persistent primitive anastomosis is not considered a concern for aneurysm formation. The persistent trigeminal artery is the most commonly observed persistent intracranial artery, with an incidence rate of up to 0.6%. It is followed by the persistent hypoglossal artery, present in 0.02–0.09% of cases, and the persistent proatlantal artery, with an incidence of 0.03%, while others, such as the otic and stapedial arteries, are exceptionally rare. Each persistent anastomosis has distinct embryological origins, anatomical variants, and potential roles in cerebrovascular compensation. Although persistent arteries may serve as collateral pathways in cases of cerebrovascular stenosis or occlusion, they can also pose risks during neurosurgical and endovascular procedures. Given their potential association with vascular pathologies in some cases, precise recognition of these arteries via imaging is essential to prevent misdiagnosis and optimise therapeutic strategies. This study provides a comprehensive review of the embryology, anatomy, and clinical relevance of persistent primitive intracranial arteries, aiming to enhance understanding, facilitate diagnosis, and improve treatment planning in cerebrovascular interventions.

Keywords: embryonic cerebral vasculature, persistent anastomoses, carotid-vertebrobasilar anastomoses

Streszczenie

Pierwotne tętnice wewnątrzczaszkowe, w tym tętnica trójdzielna, proatlantalna, uszna, podjęzykowa oraz strzemiączkowa, są embrionalnymi strukturami naczyniowymi, które tymczasowo łączą krążenie przednie i tylne lub wewnątrzczaszkowe i zewnątrzczaszkowe, zanim zwykle ulegną regresji w trakcie rozwoju. Ich przetrwanie do wieku dorosłego, choć rzadkie, może zmieniać lokalną hemodynamikę, przyczyniając się do powstawania tętniaków, zwężeń naczyń lub dysfunkcji nerwów czaszkowych. Jednak ze względu na niską częstość występowania związanych z nimi patologii sama obecność przetrwałego pierwotnego zespolenia nie jest uznawana za czynnik sprzyjający powstawaniu tętniaków. Przetrwała tętnica trójdzielna jest najczęściej obserwowaną przetrwałą tętnicą wewnątrzczaszkową, z częstością występowania sięgającą do 0,6%. Następna pod

względem częstości jest przetrwała tętnica podjęzykowa, występująca w 0,02–0,09% przypadków, oraz przetrwała tętnica proatlantalna, z częstością około 0,03%, natomiast inne, takie jak tętnica uszna i strzemiączkowa, są wyjątkowo rzadkie. Każde z tych przetrwałych zespoleń ma odrębne pochodzenie embriologiczne, warianty anatomiczne oraz potencjalną rolę w kompensacji krążenia mózgowego. Chociaż przetrwałe tętnice mogą pełnić funkcję dróg krążenia obocznego w przypadkach zwężeń lub niedrożności naczyń mózgowych, mogą również stanowić ryzyko podczas zabiegów neurochirurgicznych i wewnątrznaczyniowych. Ze względu na ich możliwy związek z patologiami naczyniowymi w niektórych przypadkach, dokładne rozpoznanie tych tętnic w badaniach obrazowych jest kluczowe dla uniknięcia błędnej diagnozy oraz optymalizacji strategii terapeutycznych. W niniejszej pracy autorzy przedstawiają kompleksowy przegląd embriologii, anatomii oraz znaczenia klinicznego przetrwałych pierwotnych tętnic wewnątrzczaszkowych, mając na celu pogłębienie wiedzy, ułatwienie diagnostyki oraz poprawę planowania leczenia w interwencjach naczyniowo-mózgowych.

Słowa kluczowe: embrionalne unaczynienie mózgu, przetrwałe zespolenia, zespolenia szyjno-kręgowo-podstawne

INTRODUCTION

Primitive intracranial arteries (PIAs), including the trigeminal, proatlantal, otic, hypoglossal, and stapedial arteries, play a significant role during the first week of embryonic development, serving as temporary connections between the developing anterior and posterior circulation. These vessels typically begin to regress around the 5th to 6th week of embryonic development, starting with the otic artery, followed by the hypoglossal, trigeminal, stapedial, and proatlantal arteries (Dumitrescu et al., 2021). Influenced by haemodynamic factors, including excessive blood flow and prolonged inadequate shear stress, endothelial cells undergo adaptive reorganisation, and primitive arteries may fail to occlude, leading to persistence into adulthood. Additionally, alterations in genes responsible for vascular remodelling, such as *PDGF* or *VCAM*, as well as epigenetic mechanisms, may contribute to defective vascular development (Nixon et al., 2010). The prevalence of PIAs into adulthood varies, with the persistent trigeminal artery (PTA) being most frequently observed, occurring in approximately 0.1–0.6% of cases (Meckel et al., 2013). The persistent hypoglossal artery (PHA) is the next most common, with an incidence rate reaching up to 0.09%, followed by the persistent proatlantal artery (PPA) occurring in up to 0.03% of the population, and the persistent stapedial artery (PSA) with a prevalence estimated at 0.01–0.02% (Govaerts et al., 1993; Srinivas et al., 2016; Tubbs et al., 2011). The persistent otic artery (POA) is exceptionally rare, reported only in isolated cases (Tubbs et al., 2011). When these arteries persist, they may alter local haemodynamics and potentially increase the risk of aneurysm formation or large-vessel occlusions (Diana et al., 2019; Meckel et al., 2013). However, no strict evidence links PIAs to vascular pathologies, as they are often incidental findings with limited clinical significance. Currently, computed tomography angiography (CTA) and magnetic resonance angiography (MRA) are the primary non-invasive methods for diagnosing PIAs. Digital subtraction angiography (DSA), although invasive, remains the gold standard for accurate and definitive assessment, and is particularly important in preoperative planning, as PIAs can be mistaken for pathological abnormalities (Pasaoglu et al., 2009). A comprehensive understanding of their anatomy and clinical implications is essential to avoid unnecessary interventions and ensure the safety of surgeries and interventional radiology.

EMBRYONIC VASCULAR DEVELOPMENT

Craniocerebral vasculature development begins around the 24th day of embryogenesis, before the onset of the heartbeat (Dumitrescu et al., 2021). By the time the embryo reaches 3 mm in length, the internal carotid artery (ICA) has formed from the third branchial arch arteries and the dorsal aorta. The first and second arches are present in the early stages of involution and are later replaced by the mandibular and hyoid arteries.

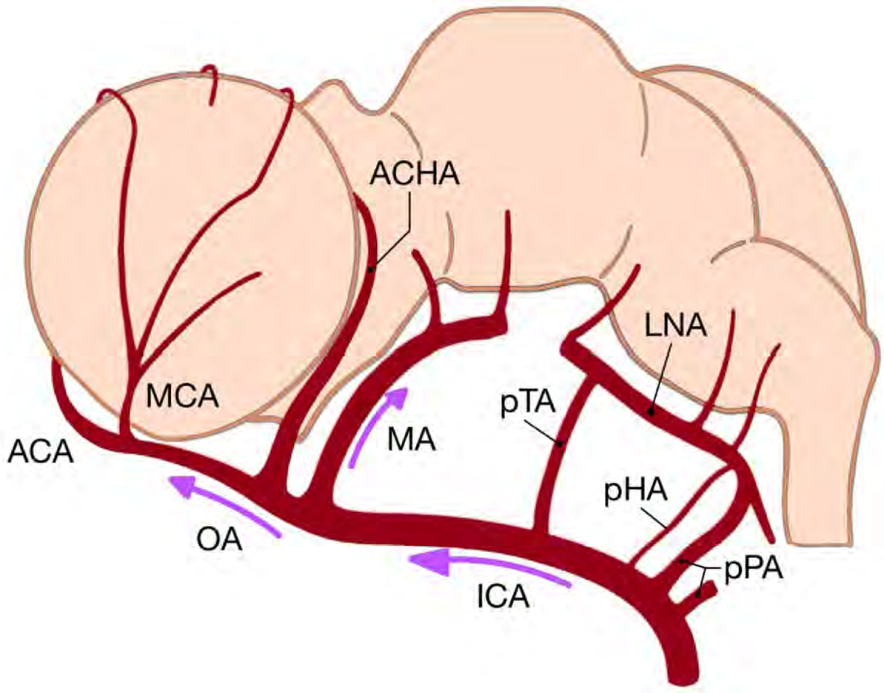
Padget defined seven stages in the development of the brain arteries, progressing from an early undifferentiated pattern to the essentially adult configuration, as described below (Padget, 1948). The carotid-vertebrobasilar anastomoses characteristic of the embryonic cerebral vasculature are illustrated in Fig. 1.

STAGE 1 (4–5 mm, 28–29 days)

The ICA supplies the forebrain, midbrain, and hindbrain vesicles. At the trigeminal ganglion, it divides into two branches. The cerebral continuation extends toward Rathke's pouch, forms the posterior circle of Willis via plexiform channels connecting to the opposite ICA, and further gives rise to the olfactory artery (OA) and the mesencephalic artery (MA). The other branch, known as the trigeminal artery, extends dorsally to join the bilateral longitudinal neural arteries (LNA), the precursors of the vertebrobasilar system. The LNA connects to the ICA via four primitive vessels: trigeminal (pTA), otic (pOA), hypoglossal (pHA), and proatlantal (pPA) arteries. These anastomoses regress once the LNA connects cranially with the ICAs and caudally with the longitudinal paravertebral anastomosis, which subsequently forms the vertebral arteries (VAs) (Padget, 1948; Raybaud, 2010).

STAGE 2 (5–6 mm, 29 days)

The caudal divisions of the ICAs extend to the mesencephalon, where they join the bilateral LNAs and become the



ACA – anterior cerebral artery; **ACHA** – anterior choroidal artery; **ICA** – internal carotid artery; **LNA** – longitudinal neural artery; **MA** – mesencephalic artery; **MCA** – middle cerebral artery; **OA** – otic artery; **pHA** – primitive hypoglossal artery; **pPA** – primitive proatlantal artery; **pTA** – primitive trigeminal artery.

Fig. 1. Schematic representation of early embryonic cerebral vasculature

posterior communicating arteries (PComAs). These vessels soon replace the primitive internal carotid system as the cranial blood supply, prompting regression of the pTA, pHA, and pOA. The pOA is the first to disappear, followed by the regression of the pHA, and finally by the obliteration of the pTA by the 12–14 mm embryonic stage (Namba, 2017). The LNAs begin to fuse along the midline to form the basilar artery (BA), but at this stage it remains dependent on supply from the pPAs (Padget, 1948; Raybaud, 2010).

STAGE 3 (7–12 mm, 32 days)

At this stage, the hyoid artery is prominent, and the cranial division of the ICA gives rise to several branches. The largest is the anterior choroidal artery (AChA), supplying the diencephalon. The anterior cerebral artery (ACA) begins to form, and its primitive branches develop into the middle cerebral artery (MCA) trunk. The ICA also gives rise to the dorsal primitive ophthalmic artery (pOpA). The posterior cerebral artery (PCA) arises initially as a continuation of the PComA to supply the mesencephalon and diencephalon. As BA fusion progresses, posterior choroidal branches (PCBs), both medial and lateral, emerge from the PCAs, along with MAs supplying the midbrain. The pTA may still be present but is usually regressing. The superior cerebellar artery (SCA) becomes distinct at the cranial end of the BA. The VAs begin to form caudally via longitudinal paravertebral anastomoses (Padget, 1948; Raybaud, 2010).

STAGE 4 (12–14 mm, 35 days)

The anterior division of the ICA now gives rise to distinct branches: the ACA, with a medial branch forming the anterior communicating artery (AComA), the early MCA, the dorsal and ventral pOpAs, AChA, PCBs, and MAs. The BA and VAs continue to develop. The hyoid artery forms a key collateral branch that passes through the stapes primordium – between the first pharyngeal pouch and the facial nerve – and connects to a distal remnant of the ventral pharyngeal artery. This branch becomes the stapedial artery (SA), which redirects selected ICA branches into the external carotid (ECA) system (Padget, 1948; Raybaud, 2010).

STAGE 5 (16–18 mm, 40 days)

With the appearance of the permanent ophthalmic artery and further differentiation of the cerebral vasculature, the adult arterial configuration becomes evident. The SA now has two major divisions: the ventral branch, forming the mandibular and maxillary (infraorbital) arteries, and the dorsal (supraorbital) branch. The SA contributes to vascularisation of the embryonic head and neck, supplying future dural, orbital, and maxillary regions. The MCA becomes a dominant stem, branching extensively across the cerebral hemisphere. Both the AChA and PCBs terminate in the choroid plexus of the lateral and third ventricles, while also supplying the mesencephalon, pineal gland, and thalamus. The future anterior inferior cerebellar artery (AICA)

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arises near the eighth cranial nerve and terminates in the choroid plexus of the fourth ventricle, while a cranial vertebral branch ascends the medulla to mark the future posterior inferior cerebellar artery (PICA) (Padget, 1948; Raybaud, 2010).

STAGE 6 (20–24 mm, 44 days) AND STAGE 7 (40 mm, 52 days)

The mature configuration of the circle of Willis is now established, with the MCA and AComA fully formed. The AICA and PICA become increasingly distinct within the vascular plexus of the caudal hindbrain. The SA regresses as its ventral and dorsal branches are incorporated into the internal maxillary and ophthalmic arteries, leaving remnants such as the caroticotympanic and superior tympanic arteries (Padget, 1948; Raybaud, 2010).

PERSISTENT TRIGEMINAL ARTERY (PTA)

The pTA, the largest embryonic carotid-basilar anastomosis, persists the longest and typically regresses around the seventh week of gestation. It usually originates from the posterolateral or posteromedial wall of the intracavernous ICA, and less commonly from the petrous ICA. The PTA is classified into two types based on its intracavernous course: lateral (petrosal) and medial (sphenoidal), with the lateral type being more prevalent (Namba, 2017). In the lateral type, the PTA arises from the posterolateral wall, courses lateral to the abducens nerve, and pierces the dura near the sensory root of the trigeminal nerve. The medial type originates from the posteromedial wall, runs medial to the abducens nerve, and pierces the dura at the dorsum sellae. After that, both types join the distal BA, typically between the AICA and SCA. The PTA may share a common origin with the meningohypophyseal trunk and can give rise to its branches, including the dorsal meningeal, tentorial, and inferior hypophyseal arteries. If it persists into adulthood, its trajectory may have significant clinical implications, such as the risk of massive haemorrhage during transsphenoidal pituitary surgery, particularly in intrasellar PTA variants (Meckel et al., 2013).

The angiographic classification of PTA was described by Saltzman (1959). In Saltzman type I, the BA proximal to the PTA is often hypoplastic, and the PComA may be absent. The PTA acts as a primary blood supply to the distal BA, PCAs, and SCAs, entering the BA distal to the AICAs but proximal to the SCAs. In Saltzman type II, or adult-type, the PTA joins the BA below the SCAs, and the PCAs are supplied by patent PComAs. The BA is fully filled by one or both VAs, making the PTA less essential (Saltzman, 1959). In Saltzman type III, one PCA is supplied by the ipsilateral PComA, while the other PCA and both SCAs are supplied by the PTA (Tyagi et al., 2020).

The clinical significance of the PTA in intracranial vascular pathologies is considered limited. While some earlier reports suggested aneurysms occur in 14–32% of PTAs, recent analyses indicate no significant increase in aneurysm risk, as the prevalence of PTA-related aneurysms is similar to that of the general population (4.2% vs. 3.7%) (Diana et al., 2019; Meckel et al., 2013). Aneurysms involving the PTA may require different therapeutic approaches depending on the Saltzman type and VA-BA anatomy. In Saltzman type I, where the VA-BA system is hypoplastic, stent-assisted techniques are preferred to avoid posterior circulation ischaemia, whereas in Saltzman type II, adequately developed VA-BA systems may allow for balloon occlusion or PTA sacrifice after confirming sufficient collateral flow (Diana et al., 2019). Rarely, the PTA contributes to large-vessel stenosis, serving either as a collateral pathway or a source of microemboli (Meckel et al., 2013). In Moyamoya disease (MMD), the PTA may act as a collateral vessel, maintaining posterior circulation during anterior circulation occlusion. However, it may also divert blood flow from the carotid artery during MCA formation, potentially promoting MMD progression. These haemodynamic alterations may contribute to the higher reported incidence of MMD in patients with PTA compared with the general population (Sun et al., 2024). The PTA is also associated with arteriovenous malformations (AVMs) and fistulas, whose presence during the embryonic period may cause haemodynamic stress, preventing PTA occlusion. The coexistence of AVMs and PTA, observed in approximately 4.5% of cases, may help maintain haemodynamic balance or serve as an access route for embolisation (Wang and Yu, 2022). Additionally, the PTA's proximity to cranial nerves may lead to dysfunction, including oculomotor and abducens nerve palsies. Its close course with the trigeminal nerve is associated with an increased incidence of trigeminal neuralgia (2.2%), caused by direct compression of the nerve root by the persistent vessel (Tyagi et al., 2020).

PERSISTENT HYPOGLOSSAL ARTERY (PHA)

The PHAs form early in embryogenesis as part of the carotid-basilar anastomoses, supplying the developing hindbrain until the vertebral and basilar arteries fully develop (Padget, 1948; Raybaud, 2010). The PHA is the second most frequently observed persistent carotid-basilar anastomosis after the trigeminal artery, occurring in approximately 0.03–0.09% of the population (Srinivas et al., 2016). It typically originates from the posterior ICA at the C1–C3 vertebral levels, though variations include origins from the CCA or ECA (Shen et al., 2019). The PHA enters the posterior cranial fossa/follows a short ascending trajectory with a slight medial and posterior curvature as it penetrates the hypoglossal canal, accompanying the hypoglossal nerve before joining the BA (Padget, 1948; Shen et al., 2019). Since arterial patency depends on sustained blood flow, a persistent PHA often coexists with hypoplastic or absent VAs and PComAs. In such cases, adequate vertebrobasilar perfusion during circulation failure often depends on the carotid system (Shen et al., 2019).

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The diagnosis of PHA relies on Lie's angiographic criteria, which define it as originating from the cervical segment of the ICA at the C1–C3 level (Lie and Hage, 1968). Since PHA frequently coexists with hypoplastic VAs and supplies the basilar territory alongside the carotid system, its identification is particularly important. Careful preservation of both anterior and posterior cerebral circulation during neurosurgical procedures is essential to prevent ischaemic complications (Jin et al., 2018). PHA may also exhibit vessel wall abnormalities, causing turbulent blood flow and exposing the BA trunk to haemodynamic stress, thereby increasing the risk of aneurysms and AVMs. A recent literature review reported an increased incidence of aneurysms linked to PHA, including basilar bifurcation aneurysms (Shen et al., 2019). Management of such vascular pathologies may be additionally challenging due to haemodynamic dependence on carotid circulation and the complex PHA anatomy, which can complicate surgical and endovascular procedures. PHA may also increase the risk of embolism in the vertebrobasilar territory, particularly in patients with carotid atherosclerosis or atrial fibrillation, as it can serve as a pathway for emboli from carotid plaques to the posterior circulation (Jin et al., 2018; Srinivas et al., 2016). Although anterior and posterior circulation infarcts linked to PHA are extremely rare, the potential for simultaneous infarcts in both territories should not be overlooked (Jin et al., 2018).

PERSISTENT PROATLANTAL ARTERY (PPA)

The PPA may originate from the common carotid artery (CCA, 5%), ECA (58%), or ICA (38%) at the C2–C4 level (Ma et al., 2019). It joins the VA in the suboccipital region and traverses the foramen magnum. The main types of PPA are classified based on origin and trajectory. Type I originates from the posterior ICA or distal part of CCA, courses between the C1 arch and the occiput, and enters the foramen magnum to connect with the VA. It is considered a remnant of the C1 segmental artery. Type II arises from the proximal ECA, follows a lateral course through the C1–C2 space, and joins the VA after passing through the C1 transverse foramen. This type is considered a persistence of the C2 segmental artery (Bour et al., 1991; Lasjaunias et al., 1978). A variant, described as a "mixed PPA", combines features of both types, originating like type II from the ECA but bypassing the cervical vertebra transverse foramina and following a type I trajectory, directly joining the VA and entering the foramen magnum. Angiographically, the PPA is often confused with the PHA, but differentiation is possible in the lateral view at the suboccipital region, where the PPA follows a trajectory similar to that of a normal VA (Ma et al., 2019).

Up to 59% of patients with a PPA have vascular anomalies, most commonly ipsilateral or bilateral VA absence or hypoplasia, occurring in approximately 50% of cases. These anomalies disturb blood flow and increases the risk of ischaemic stroke in the posterior vascular area. Other associated anomalies include aneurysms, AVMs, carotid-basilar anastomoses, vein of Galen malformations, and aortic arch abnormalities, indicating the role of haemodynamic factors involving the PPA in cerebrovascular pathologies (Ma et al., 2019). In cases of significant carotid or vertebral artery stenosis, the PPA often compensates via anastomoses between the occipital and vertebral arteries, which can present a therapeutic dilemma due to the risk of posterior cerebral ischaemia during endovascular approach (Bour et al., 1991; Namba, 2017). It may also serve as a potential source of thromboembolism, particularly during embolisation procedures, requiring careful preoperative evaluation to minimise complications (Bour et al., 1991). Recognition of this persistent anastomosis in treatment planning is important for the establishment of adequate blood flow in patients with cerebral vessel diseases, such as acute BA occlusion or ICA stenosis.

PERSISTENT STAPEDIAL ARTERY (PSA)

The prevalence of PSA is estimated at 0.01–0.02% in the general population (Govaerts et al., 1993). Four primary anatomic variations have been described in the literature: hyoidostapedial artery, pharyngostapedial artery, persistent stapedial artery with pseudo-petrous ICA, and pharyngohyostapedial artery. The most common is the hyoidostapedial artery, where the SA trunk persists postnatally, resulting in an absent foramen spinosum and the MMA originating directly from its trunk. A rarer but clinically significant variant, the PSA with pseudo-petrous ICA, is characterised by the absence of the carotid foramen and cervical ICA, replaced by a compensatory pathway in which the inferior tympanic artery supplies the intrapetrous ICA through the PSA. This configuration increases the risk of iatrogenic injury to compensatory vessels, potentially leading to haemorrhage, ischemia, or cranial nerve damage. Other anatomic variants have been documented in a limited number of cases (Hitier et al., 2013).

Most PSA cases are incidental, though some present with conductive hearing loss or pulsatile tinnitus. Conductive hearing loss likely results from ossicular dampening by the PSA or stapes malformations. Pulsatile tinnitus is thought to arise from sound transmission through the cochlear promontory, and possibly from stapedial pulsations if the PSA contacts the stapedial crura. The PSA has been also linked to congenital abnormalities and chromosomal conditions, including trisomies 3, 13, 15, and 21, Paget disease, congenital cholesteatoma, neurofibromatosis, and immunodeficiency syndromes. Therefore, the identification of a PSA necessitates thorough assessment for potential associated abnormalities (LoVerde et al., 2021).

PSA poses challenges for otologic and neurosurgical procedures due to its complex course, anatomical variations, and risk of otoneurological complications (Hitier et al., 2013). In rare cases, a PSA may accompany an aberrant ICA emerging in the hypotympanum without a protective bony

canal, making it vulnerable to injury during surgery (Govaerts et al., 1993). Iatrogenic PSA injury can cause bleeding, facial hemiparesis, hemiplegia, and auditory or vestibular disturbances (LoVerde et al., 2021). However, untreated symptomatic PSA and stapes abnormalities often lead to persistent conductive hearing loss and pulsatile tinnitus, impairing long-term quality of life. Surgical management of PSA includes three approaches: stapedectomy with preservation of the PSA, PSA transection without stapedectomy, and a combined approach. While stapes surgery effectively treats conductive hearing loss, its impact on pulsatile tinnitus remains uncertain. However, PSA transection alone has proven effective for alleviating pulsatile tinnitus. The high prevalence of footplate ankylosis in symptomatic PSA further complicates surgery, often necessitating stapes procedures and making footplate mobilisation more challenging (Goderie et al., 2017).

PERSISTENT OTIC ARTERY (POA)

The POA, first identified in 1969 in an autopsy specimen, has since been reported in only a few cases. Its identification remains challenging, as most reports lack anatomical validation and rely on lateral skull imaging, which poorly delineates its relationship to bony landmarks (Patel et al., 2003). During embryonic development, the primitive otic artery (pOA), along with three other carotid-basilar anastomoses, temporarily connects the ICA to the bilateral LNAs, contributing to hindbrain vascularisation and BA formation (Tubbs et al., 2011; Namba, 2017). The pOA emerges from the ICA within the carotid canal near the small acoustic sac, passes through the internal acoustic meatus, and joins the BA at its caudal end (Lie and Hage, 1968). Its early obliteration likely accounts for its rarity compared with other persistent carotid-basilar anastomoses (Patel, et al., 2003).

The clinical relevance of the POA lies in its potential to complicate endovascular or surgical treatment, particularly when associated with aneurysms or AVMs. Given its role in connecting the ICA to the BA, the POA may exacerbate blood flow disruptions and worsen posterior circulation infarcts. Its location within the internal acoustic meatus places it adjacent to the facial and vestibulocochlear nerves, potentially causing compression-related symptoms (Vasović et al., 2010). The presence of a POA may be also associated with other cerebral vascular anomalies, such as hypoplasia or aplasia of the VAs or PComAs, further complicating neurosurgical procedures (Tubbs et al., 2011).

CONCLUSIONS

Primitive intracranial arteries, though rare, play a critical role in cerebrovascular pathologies and require precise identification to prevent complications during treatment. Their persistence may contribute to compensatory haemodynamics or exacerbate vascular anomalies, including IAs, AVMs, or cerebral vessel stenosis. Understanding PIAs is essential for accurate diagnosis, safe surgical planning, and targeted therapeutic interventions, highlighting the need for increased awareness and further research into their clinical implications.

Conflict of interest

The authors do not report any financial or personal connections with other persons or organisations which might negatively affect the content of this publication and/or claim authorship rights to this publication.

Author contribution

Original concept of study: NAK, KW. Collection, recording and/or compilation of data: NAK, PO. Analysis and interpretation of data: NAK, PK, AD, MP, KZ. Writing of manuscript: NAK, PO, AD, KW. Critical review of manuscript: MP, KZ, KW, DJ. Final approval of manuscript: NAK, PO, AD, MP, KZ, KW, DJ.

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