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ORIGINAL ARTICLE

Rare congenital anomalies of the internal carotid artery: anatomic and radiologic aspects of three cases and review of the literature

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Abstract

Purpose Congenital anomaly of the internal carotid artery (ICA) is a rare entity. It is usually discovered incidentally by color doppler carotid sonography, angiography, computerized tomography (CT), or magnetic resonance imaging of the head and neck region taken for some other reasons. The aim of this study was to detect congenital ICA anomalies, to delineate existing collateral vessels and to find out its incidence.

Methods 1847 patients' CT angiography images of the head and neck region taken between May 2013 and February 2018 were retrospectively evaluated for ICA anomalies.

Results We detected three cases (0.16%) with unilateral agenesis of ICA, bilateral agenesis of ICA and bilateral hypoplasia of ICA, respectively. Most patients are asymptomatic because of collateral cerebral circulation supplied by the communicating arteries of the circle of Willis, intercavernous anastomosis, communicating arteries from the external carotid artery, and by persistent embryologic arteries to the carotid artery territory.

Conclusion Recognition of ICA anomalies has important implications during planned carotid or transsphenoidal surgery, in thromboembolic disease, and in the follow-up and detection of associated cerebral aneurysms.

Keywords Internal carotid artery · Agenesis · Hypoplasia · CT angiography

Background

Congenital anomaly of the internal carotid artery (ICA) is a rare entity [4, 22]. It is usually discovered incidentally by color doppler carotid sonography, angiography, computerized tomography (CT), or magnetic resonance imaging (MRI) of the head and neck that are taken for some other reasons [22, 26]. The prevalence of the ICA anomalies has been reported to be around 0.01% in the literature [24, 25].

According to Lasjaunias et al. [12], the different segments of ICA arise from different embryologic segments, specifically the following arteries: ventral pharyngeal, hyoid, mandibular, primitive maxillary, trigeminal, dorsal ophthalmic, and ventral ophthalmic. Agenesis of the ICA occurs due to abnormal regression of the first and third aortic arch, with the exact etiology being unclear.

The terms agenesis, and hypoplasia are often misused in the setting of an absent or small ICA. Lie and Hage [17] defined agenesis as the total absence of ICA, whereas hypoplasia refers to cases where a segment or whole length of the ICA is narrower. For this reason, the bony carotid canal is absent in agenesis, whereas it is present but narrower in hypoplasia.

Most patients are asymptomatic because of collateral cerebral circulation supplied by the circle of Willis, intercavernous anastomosis, external carotid artery (ECA), and persistent embryologic arteries to the carotid artery territory [18]. Sometimes they live without any symptom during their whole life [19].

Head and neck CT angiography of three cases were reported by an expert neuroradiologist (RG) and an anatomist (AF) as unilateral agenesis of ICA, bilateral agenesis of ICA and bilateral hypoplasia of ICA. The aim of this study was to report these cases with characteristic anatomic and

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radiologic aspects in patients to delineate existing collateral vessels.

Materials and methods

Head and neck CT angiography images of 1847 patients (1240 female, 607 male) performed between May 2013 and February 2018 were included in this study. Mean age of patients was 61 years. A 64–detector row dual CT scanner (Somatom Definition, Siemens Healthcare, Erlangen, Germany) was used for CT imaging. Only arterial phase imaging was performed. The protocol was as follows: 64 × 0.6 collimation, 1.4 pitch, 0.5-s rotation time, 100 kV (peak) and with 180 effective mAs. The source images were reconstructed into 1-mm slice thicknesses in axial view, and coronal and sagittal images were reformatted. It is a standard procedure in our health center that we obtain an informed consent from all patients who undergo CT angiography imaging where we use a contrast agent.

All the images were reviewed by an expert neuroradiologist (RG) and an anatomist (AF). There was consensus among the experts for diagnosis of each case.

Results

We detected three cases (0.16%) with congenital ICA anomalies. The cases are as follows: unilateral ICA agenesis, bilateral ICA agenesis and bilateral ICA hypoplasia.

Case 1

A 34-year-old woman presented with headache that lasted for 3 days and relieved after taking analgesic medication. For further evaluation, CT angiography of the head and neck region was performed, and images indicated a normal course of right common, internal, and external carotid arteries. However, coronal CT angiography showed a narrow left common carotid artery followed by a single vessel with the course of left ECA (Fig. 1a).

Axial CT section of the skull base showed the absence of left carotid canal. Right carotid canal was intact and was examined in normal caliber (Fig. 1b). This finding categorizes our case as left-sided ICA agenesis.

3D volume rendering of intracranial vasculature from CT angiography confirmed the agenesis of left ICA and showed the abnormal vessel originating from the cavernous segment of right ICA and reaching the left supraclinoid area. Then, this abnormal vessel united with the origin of the left middle cerebral artery (MCA). Left MCA was supplied by this abnormal anastomotic vessel and left patent posterior communicating artery (PCOM). The caliber of the left MCA was normal. A2 segment of the left anterior cerebral artery (ACA) was supplied by anterior communicating artery (ACOM). A1 segment of left ACA and right PCOM could not be seen. The intracranial vertebrobasilar system was normal (Fig. 1c). This is type D collateral formation according to Lie and Hage's study

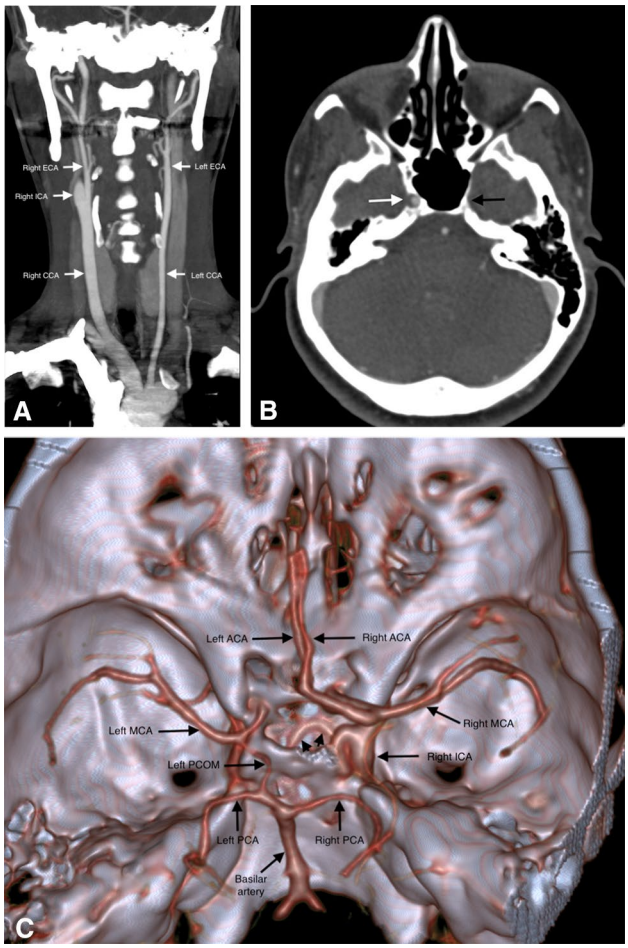

Fig. 1 **a** Coronal reformatted CT angiography image shows the normal course of the right CCA, ICA and ECA. Left CCA is thin and continuing as only the ECA; however, the left ICA is absent. (*CCA* common carotid artery, *CT* computerized tomography, *ECA* external carotid artery, *ICA* internal carotid artery). **b** Axial CT section at the level of skull base demonstrates the absence of the left carotid canal (black arrow) and a normally developed right carotid canal (white arrow). (*CT* computerized tomography). **c** 3D volume rendering of intracranial vasculature from CT angiography demonstrates the absence of left ICA and an abnormal vessel (black arrowheads) originating from the cavernous segment of right ICA and supplying the left MCA at the supraclinoid level. Left MCA is supplied by both this abnormal anastomotic vessel (intercavernous anastomosis) and patent left PCOM. The caliber of left MCA is normal. A2 segment of left ACA is supplied by ACOM. A1 segment of left ACA and right PCOM are absent. The vertebro-basilar system seems normal. (*CT* computerized tomography, *ACA* anterior cerebral artery, *ICA* internal carotid artery, *MCA* middle cerebral artery, *PCA* posterior cerebral artery, *PCOM* posterior communicating artery)

[17]. Type D, also known as transsellar anastomosis, is extremely rare.

Case 2

A 43-year-old man admitted to the hospital with dizziness. Carotid doppler ultrasonography was performed and bilateral absence of ICA was detected. Coronal CT angiography of the head and neck region indicated normal course of the bilateral common carotid arteries. But there were no bifurcation and common carotid arteries prolonged with the ECAs bilaterally (Fig. 2a). Bilateral ICA agenesis was shown on the 3D volume rendering of intracranial vasculature from CT angiography (Fig. 2b).

At the level of the skull base, both carotid canals were absent, indicating bilateral ICA agenesis (Fig. 2c).

Both MCAs and ACAs were filled from the enlarged PCOMs via the circle of Willis. MCAs had normal caliber and a normal distal branching pattern bilaterally. ACOM could not be seen (Fig. 2d). Bilateral vertebral arteries were enlarged and measured approximately 6 mm in diameter (Fig. 2e). The origins of the left subclavian artery and

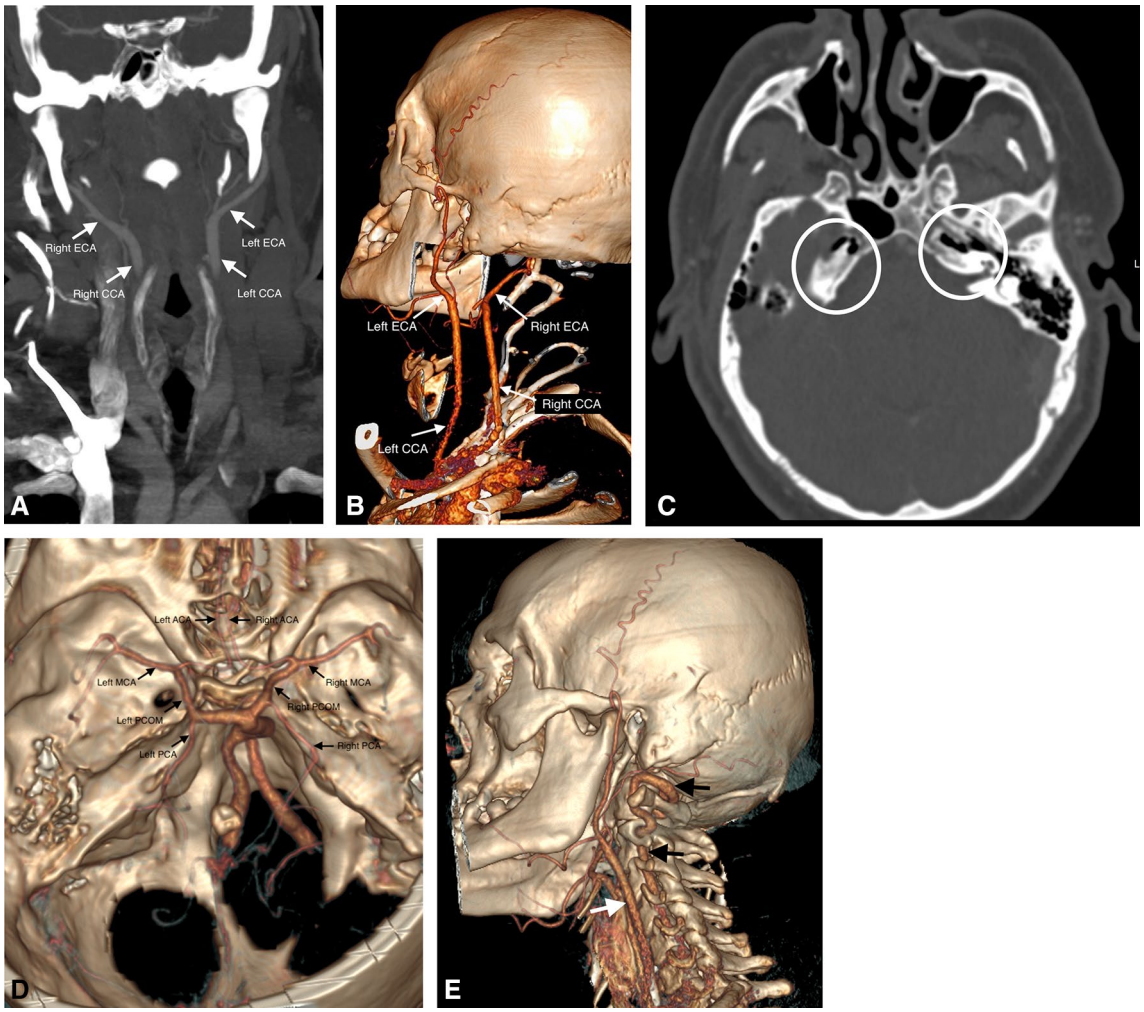

Fig. 2 **a** Coronal reformatted CT angiography image shows the normal course of the bilateral CCAs. But there are no bifurcation and CCAs are continuing as the ECAs bilaterally. (*CCA* common carotid artery, *CT* computerized tomography, *ECA* external carotid artery). **b** 3D volume rendering of intracranial vasculature from CT angiography demonstrates the absence of the bilateral ICA. (*CCA* common carotid artery, *CT* computerized tomography, *ECA* external carotid artery, *ICA* internal carotid artery). **c** Axial CT section at the level of skull base shows bilateral absence of bony carotid canals (white circles). (*CT* computerized tomography). **d** 3D volume rendering of intracranial vasculature from CT angiography reveals the absence of the bilateral ICA. Both MCAs and ACAs are supplied by the large PCOMs. MCAs have normal caliber and a normal distal branching pattern bilaterally. There is no ACOM. Bilateral vertebral arteries are seen enlarged. (*CT* computerized tomography, *ACA* anterior cerebral artery, *ICA* internal carotid artery, *MCA* middle cerebral artery, *PCA* posterior cerebral artery, *PCOM* posterior communicating artery). **e** 3D volume rendering of intracranial vasculature from CT angiography shows the normal course of left CCA (white arrow) and the enlarged left vertebral artery (black arrows). (*CCA* common carotid artery, *CT* computerized tomography)

brachiocephalic trunk were seen ectatic. This is type C collateral circulation according to the Lie and Hage's classification [17].

Case 3

A 27-year-old woman presented with suspected vasculitis. Head and neck CT angiography showed normal course and caliber of common carotid arteries, but after bifurcation point, bilateral ICAs were measured only 2 mm in diameter at the level of C3 cervical vertebrae (Fig. 3a).

Axial CT section showed bilateral hypoplastic ICAs along with their courses (Fig. 3b).

Axial CT section through the skull base indicated smaller carotid canals bilaterally, which corresponded in size to the hypoplastic ICAs (Fig. 3c).

Both MCAs and ACAs were filled from the enlarged PCOMs via the circle of Willis. MCAs had normal caliber and a normal distal branching pattern bilaterally. A1 segment

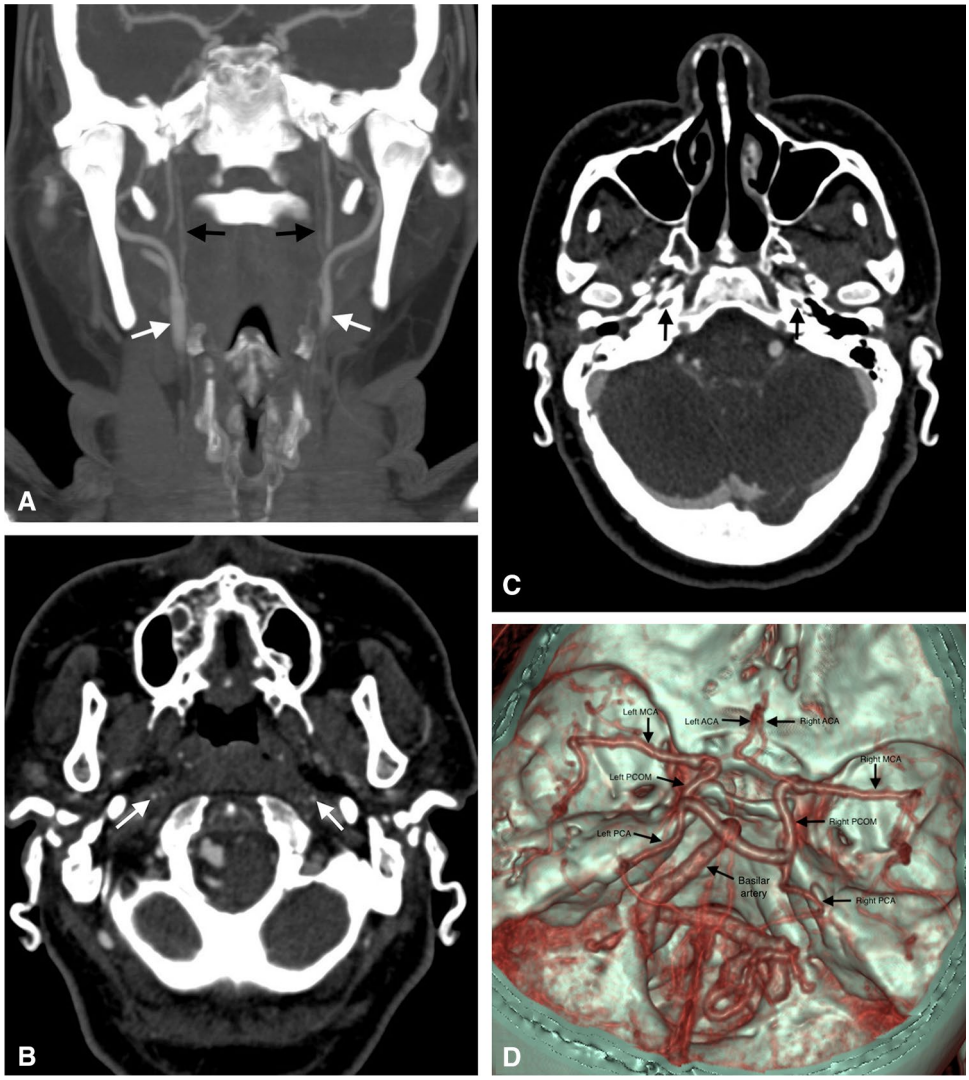

Fig. 3 **a** Coronal reformatted CT angiography shows the normal course of CCAs (white arrows) and very thin (hypoplastic) ICAs (black arrows). (*CCA* common carotid artery, *CT* computerized tomography, *ICA* internal carotid artery). **b** Axial CT section shows bilateral hypoplastic ICAs (white arrows). (*CT* computerized tomography, *ICA* internal carotid artery). **c** Axial CT section of the skull base shows bilateral hypoplastic carotid canals (black arrows). (*CT* computerized tomography). **d** 3D volume rendering of intracranial vasculature from CT angiography shows that both MCAs and ACAs are filled from the enlarged PCOMs via the circle of Willis. MCAs have normal caliber and a normal distal branching pattern bilaterally. A1 segment of right ACA has normal caliber, but left A1 segment is detected hypoplastic. There is no ACOM. The caliber and course of the left vertebral artery are normal, but the right vertebral artery is observed tortuous and ectatic along its course. In addition, the right vertebral artery has focal fusiform aneurysms. (*CT* computerized tomography, *ACA* anterior cerebral artery, *MCA* middle cerebral artery, *PCA* posterior cerebral artery, *PCOM* posterior communicating artery)

of right ACA had normal caliber, but left A1 segment was detected hypoplastic. On CT sections, ACOM could not be seen (Fig. 3d). The caliber and course of the left vertebral artery were normal, but the right vertebral artery was observed tortuous and ectatic along its course. Focal fusiform aneurysms were detected at the intradural part of the right vertebral artery. The collateral circulation is type E according to the classification of Lie and Hage [17].

Discussion

The paired ICAs, two of the four major arteries that supply the brain, provide most of the blood flow to the cerebral hemispheres [20]. The estimated prevalence of congenital ICA anomalies is around 0.01% in the literature [4, 22, 24, 25]. Recognition of pre-existing ICA anomalies has crucial implications during planned carotid or transsphenoidal surgery, in thromboembolic disease and in the follow-up and detection of associated cerebral aneurysms [4]. It has been vital importance to be aware of vascular anomalies such as aneurysm or arteriovenous malformation in ICA anomalies if an interventional radiological treatment is needed [3]. In our study, we detected three patients (0.16%) with abnormal ICA among 1847 CT angiography images. This finding is higher than the literature knowledge. This higher result may be because such rare and complicated cases were referred to third-level hospitals in our country. Ito et al. [11] reported that they examined 1275 angiography series and found three cases (0.24%) with congenital ICA anomalies: unilateral agenesis in one and unilateral hypoplasia in two. Taşar et al. [26] evaluated 5100 cerebral MRI and/or catheter angiograms and observed seven patients (0.13%) with congenital ICA anomalies (agenesis in three and hypoplasia in four patients). The prevalence of ICA anomalies found by Ito et al. [11] and Taşar et al. [26] was higher, as in our study.

There are at least 33 reported cases with bilateral ICA agenesis in the literature [2]. Bilateral ICA segmental agenesis is an extremely rare congenital anomaly and it was reported in 2016 by Alexandre et al. [2]. They stated that an 18-year-old man admitted to the hospital with occasional mild headaches and the result of his MRI showed bilateral agenesis of the cervical segments of ICAs. Left common carotid injection showed that the petrous segment of left ICA opacifies through anastomotic vessels arising from the internal maxillary artery. They showed that the intracranial ICA and ipsilateral PCOM were dolichoectasic. Right common carotid injection visualized the right ICA only from the distal part of the ophthalmic segment and opacified by rich anastomotic vessels through ipsilateral internal maxillary artery. Our case with bilateral ICA agenesis had enlarged PCOMs and vertebral arteries on each side for supplying MCAs and ACAs bilaterally.

The differential diagnosis between congenital and acquired pathologies of ICA is crucial for clinicians. Agenesis and hypoplasia can be distinguished from acquired ICA occlusion or stenosis by examining the carotid canal on CT images. The bony carotid canal is absent in agenesis, whereas it is present but narrower in hypoplasia. Normal-sized bony carotid canal is found in acquired ICA occlusion or stenosis [5, 9, 20, 22]. Acquired stenosis of ICA lumen may develop secondary to arteriosclerosis, arteritis, fibromuscular dysplasia, intimal dissection or Moya-Moya diseases [26]. In acquired ICA occlusion or stenosis, perfusion of cerebral hemispheres is often reduced and patients often present with a stroke, whereas patients with congenital ICA anomalies are commonly asymptomatic for long periods [11].

Carotid agenesis can be either unilateral or bilateral, with a higher proportion of cases being unilateral. There is also a predilection to the left-sided carotid agenesis, with a reported ratio of cases 3:1. However, Lee et al. [14] stated nine patients with congenital ICA anomalies and the ratio of right/left side was found 5:3, in contrast to the literature knowledge. The true prevalence of ICA agenesis is unclear, with most cases found incidentally by ultrasound, MRI, or CT. Most cases are clinically silent, due to well-developed collateral circulations [16]. The circle of Willis, intercavernous anastomosis, persistent trigeminal arteries, persistent tympanic or stapedial arteries, hypoglossal artery, ophthalmic arteries and ECAs were described as collateral pathways in the literature [6, 9, 10, 19, 22]. It has been determined in our study that the vertebrobasilar system via the circle of Willis is the main collateral pathway in bilateral ICA agenesis or hypoplasia, similar to Taşar's et al. work [26]. The incidence of a persistent trigeminal artery as a collateral pathway published in the literature ranges between 0.1 and 0.6% [1, 7, 8]. Pilleul et al. [22] stated that CT angiography of a 18-year-old man showed bilateral ICA agenesis and detected anastomoses between the carotid and vertebro-basilar system via a patent primitive trigeminal artery. Mellado et al. [18] reported that a 29-year-old woman with a history of congenital hypopituitarism was diagnosed unilateral agenesis of ICA with a trans-sellar anastomosis. In one of our cases (unilateral ICA agenesis), trans-sellar anastomosis was detected similarly. Hong et al. [10] noticed that a 69-year-old man admitted to the hospital with a memory disturbance and he was diagnosed unilateral ICA agenesis with a rare collateral pathway, via supraclinoid-supraclinoid anastomosis.

The etiology of congenital ICA anomalies is still unknown. Some authors have claimed that agenesis of the ICA occurs due to abnormal regression of the first and third aortic arch [12]. Lasjaunias and Santoyo-Vazquez [13] indicated that seven different embryological segment branches come together and form the ICA system. Those are cervical

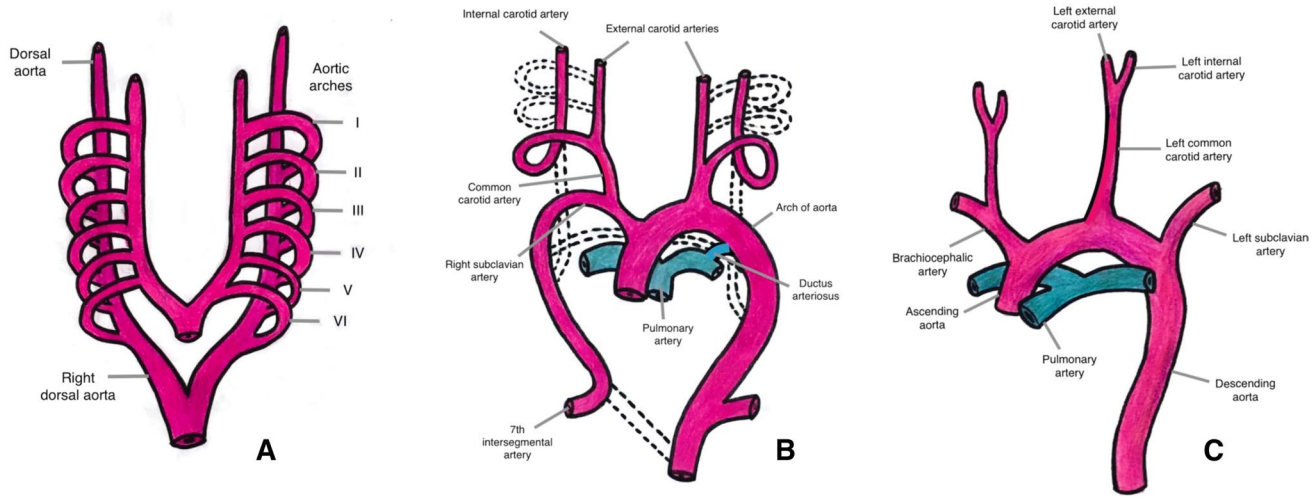

Fig. 4 Schema of embryological development of the aortic arches and carotid arteries. This illustration was inspired by the description of embryological development of cardiovascular system [13] [23]. **a** Overview of the aortic arches and dorsal aorta. **b** After regression of many arches and vessels (broken lines). **c** Mature aortic arch and common carotid arteries

segment from the third aortic arch, ascending intrapetrous segment from the second and third aortic arch, horizontal petrous segment from the first and second aortic arch, ascending segment in foramen lacerum from the first aortic arch and primitive maxillary artery, horizontal segment of carotid siphon from the primitive maxillary artery and dorsal ophthalmic artery, clinoid segment from the dorsal and ventral ophthalmic artery and finally terminal segment from the primitive ophthalmic artery and ACA (Fig. 4) [23]. Lack of development of one or more those branches may eventually lead to anomalies of ICA.

Lie and Hage [17] described six pathways of collateral circulation in association with agenesis or hypoplasia of the ICA. In **Type A**, unilateral agenesis of the ICA is associated with collateral circulation to the ipsilateral ACA through a patent ACOM and to the ipsilateral MCA from the posterior circulation through a hypertrophied PCOM. In **Type B** pattern of collateral flow, the ipsilateral ACA and MCA are supplied across a patent ACOM. **Type C** represents bilateral agenesis of the ICA with supply to the anterior circulation via carotid-vertebrobasilar anastomoses. **Type D** represents unilateral agenesis of the cervical portions of the ICA with an intercavernous communication to the ipsilateral carotid siphon from the contralateral cavernous ICA. In **Type E**, tiny ACAs are supplied by bilateral hypoplastic ICAs and MCAs are supplied by enlarged PCOMs. The **Type F** pattern provides collateral flow to the distal ICA via transcranial anastomoses from the internal maxillary branches of the ECA system (Fig. 5). When we examined our cases in terms of collateral pathways, we found that cases 1, 2 and 3 were consistent with type D, type C and type E, respectively.

There are also some clinical symptoms which have been reported to be associated with ICA agenesis include pulsatile tinnitus, ischemic stroke, transient ischemic attack symptoms, intracranial hemorrhage, symptomatic epilepsy, migraine and Horner's syndrome [3, 15, 27]. Sunada and Inoue [25] reported the first case of bilateral ICA agenesis presented with intracerebellar hemorrhage. Zink et al. [28] found 27.8% of cases involving carotid agenesis or hypoplasia associated with intracerebral aneurysms, compared to a incidence of 2–4% in the general population. Lee et al. [14] observed nine patients with congenital ICA anomalies and six of them had cerebral aneurysms. Four of them had aneurysms located on ACOM. Increased hemodynamic pressure in the collateral arteries of the dysplastic vessels is known to cause the aneurysms. CT and MRI always have to be included in the radiologic agenda of the clinician for imaging of those vessels [26]. Hypoplasia of the ICA has been reported both as an isolated anomaly and in conjunction with other abnormalities such as anencephaly and basal telangiectasia [21].

As a conclusion, congenital anomalies of the ICA are rare entities. Patients are usually asymptomatic due to collateral cerebral circulation. Detection of ICA anomalies is important before carotid or transsphenoidal surgeries, in thromboembolic disease and in the detection of associated cerebral aneurysms.

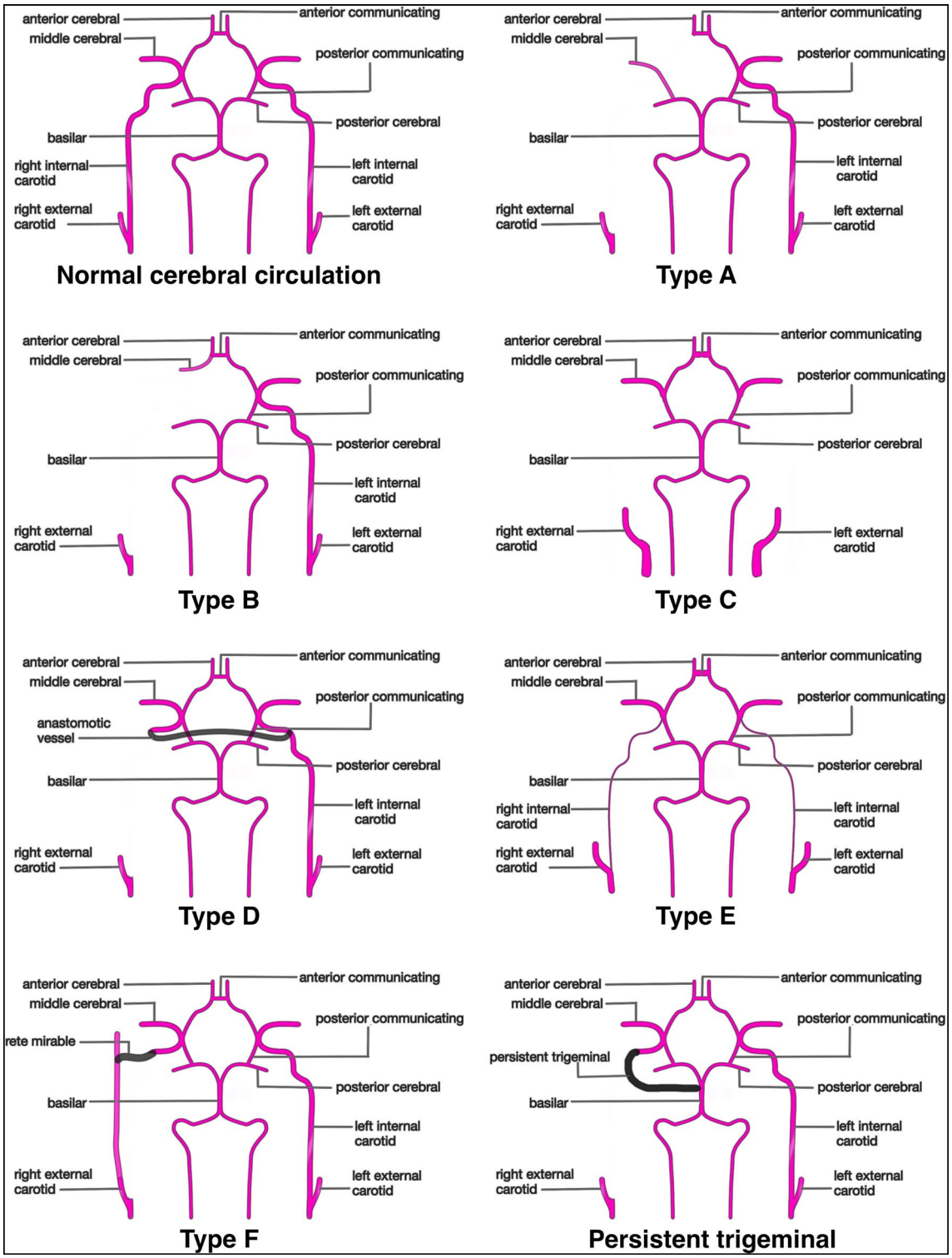

Fig. 5 Schema of the different types of arterial anastomosis in congenital ICA anomalies. This illustration was created according to the Lie and Hage's classification in 1968 [16, 17]

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Compliance with ethical standards

Conflict of interest The authors have no conflict of interest to declare.

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