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ANTERIOR CEREBRAL–ANTERIOR COMMUNICATING COMPLEX IN THE POSTNATAL PERIOD: FROM A FENESTRATION TO THE MULTIPLICATION OF ARTERIES

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Abstract. *The anterior cerebral artery is a medial terminal branch of the cerebral part of the internal carotid artery on both sides. These paired arteries are connected by the anterior communicating artery. Different abnormalities of the anterior communicating–anterior cerebral arteries can include an aplasia or hypoplasia, or variable origin and/or course and/or termination, or fenestration, or duplication or multiplication or persistence of primitive or additional vessels. The aim of this manuscript was to investigate the relationships of morphological abnormalities of an anterior communicating–anterior cerebral complex in human adults of Serbian population. Material represented 266 human cadavers autopsied at the Institute for Forensic Medicine in Niš. Cerebral arteries were investigated macroscopically, under the glass; outer vessel's diameter was measured using ImageJ processing program. A total of 87 cases or 32.71% of different abnormalities of the anterior communicating–anterior cerebral complex were found and classified into six groups. The group of duplications of the anterior communicating artery with an incidence of 18.04% was the most frequent. The finding of only four aneurysms on the anterior communicating artery indicates that there was no significant difference in the rate of aneurysms in individuals of Serbian population with and without fenestrations or duplications or multiplications of the anterior cerebral–anterior communicating complex.*

Key words: *Human adult, brain base, anterior cerebral artery, anterior communicating artery, abnormalities*

Introduction

The anterior cerebral artery (ACA) is one of two terminal branches of the internal carotid artery (ICA) on the human brain base. The ACA usually courses anterior and medially to the interhemispheric fissure and passing over the optic chiasm and nerves it joins at the midline the opposite one through the anterior communicating artery (ACoA) [1]. There are two topographical parts of the ACA – precommunicating (A1) and postcommunicating (A2) segments regarding the point of ACA–ACoA junction [2]. The communicating and choroid subparts of the cerebral part of ICA with A1 segments and ACoA represent vascular components of an anterior segment of the cerebral arterial circle (CAC) [3].

The left and right A1 segments varied in diameter from 0.9 to 4.0 mm in USA population [4] or from 2.5 to 3.5 mm in Indian population [5], and in length from 7.2 to 18.0 mm [4], or from 10 to 19 mm [5], sending two to 15 perforating arterioles [4]. These (posteroinferior and posterosuperior) arterioles usually supplied the anterior cerebral commissure and globus pallidus, the optic chiasm, the anterior perforated substance, the genu of the internal capsule, the anterior hypothalamus and part of the thalamus [5].

Unpaired ACoA was commonly between 0.2–3.4 mm in caliber in USA population [4], or between 1.0 to 4.0 mm in Indian population [5], whereas it was between 0.3–7.0 mm in length [4]. Their branches (up to four) supplied the optic nerves and chiasm, suprachiasmatic area, anterior perforated substance, lamina terminalis and frontal lobe [4].

The variants and abnormalities of arteries can include an aplasia or hypoplasia, or variable origin and/or course and/or termination, or fenestration, or duplication or multiplication or persistence of primitive or accessory vessels, etc. There were anatomical descriptions of some of the ACA and ACoA abnormalities [3–12] and/or their branches [1,3–7,11]. Majority of authors reported simultaneous presence of fenestrations and (in)direct aneurysms of the ACA–ACoA complex [1,7,13–30], or arteriovenous malformation [23], but usually in case reports.

Our aim was to describe a relationship of presented fenestration and/or duplication and/or multiplication with pathological states of the ACoA and ACA in A1 and proximal A2 segments in human adult specimens of Serbian population.

Material and Methods

We defined the ACoA and ACA in A1- and neighboring subpart of the A2 segment as an ACA–ACoA complex

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according to Pai et al. [5]. These vessels were explored on the brain base of 266 adult human cadavers of both genders autopsied at the Institute of Forensic Medicine in Niš because of different causes of death. The co-author (MT) investigated cerebral arteries during her academic and doctoral studies in accordance with the approval granted by our Ethics Committee (no. 01-206-1).

After the extraction of the brain from the cranium, each brain base with a ruler on it was photographed before and after the removal of the arachnoidea mater. Vessels' outer diameter was calculated using ImageJ processing program (http://rsb.info.nih.gov/ij/index.html). General data regarding the cadaver (cause of death, gender and age) and visible morphological abnormalities (additional vessels, partial or total duplication and triplication and/or fenestration) or associated aneurysm of the ACA and/or ACoA were noted for each case.

The criteria in defining the arterial abnormalities were according to the previous investigation [31]. A fenestration was defined as an abnormality that is characterized by a common origin of an artery that splits into two parallel channels, which subsequently rejoins into the same artery. Partial duplication of the artery was determined if it had a common origin on one and two separate junctions on the opposite artery. Two independent vessels' origin and termination characterized their total duplication. Three independent vessels or partially duplicated artery associated with the same single vessel characterized a triplication. A new term — anterior communicating network or the rete communicans anterior was a personal proposal for a special shape of the ACoA — a presence of two or more transverse ACoAs and at least of one vertically or obliquely oriented vessel among them. The drawings of ACoA abnormalities were modified according to Kayembe et al. [14].

Hypoplastic caliber (≤ 1.0 mm) of vessels was noted if it was only found in the group of investigated abnormalities; atheromatous plaques were noted if they were found within fenestration limbs or double or multiple vessels.

Results

We found 87 cases (32.71%) — 45 of male (25 to 90 years old) and 42 of female gender (16 to 95 years old) of single and/or associated abnormalities of the ACA–ACoA complex (Fig. 1).

Special morphological abnormalities of this complex were described within six groups:

I. *Arterial fenestration.* There were 13 cases (9 of female and 3 of male gender) of ACA fenestrations (Fig. 2). There were four cases of the single A1 fenestration (two on the right and two on the left) and six cases (five on the right and one on the left) of associated abnormalities and/or pathologies of the ACA–ACoA complex, as well as one case of single fenestration of the left A1–A2 segment and two cases (on the right and left) of its association with other abnormalities of the ACA–ACoA complex. All fenestrations were oval in the shape; a medial limb of the fenestration was smaller in six specimens, whereas a lateral limb was two times smaller. The perforating arterioles from medial limbs were constant, except in one specimen (case 34). Large fenestrations were found three times on the left (cases 23, 47 and 53) and once (case 68) on the right side. The ACAs with fenestrations were of normal outer diameter.

Associated abnormalities were as follows: 1) rete communicans anterior (two cases); 2) duplication of the ACoA with caudally hypoplastic vessel (four cases); 3) aplasia of the left ACA and simultaneous double fenestrations of a proximal part of the right ACA (one case); and 4) persistent right primitive olfactory artery (one case). Pathological states were as follows: insular atheromatous plaques in three cases and diffuse atheromatous plaques in fenestration limbs in one case were found, as well as one simultaneous presence of the ACoA aneurysm (case 28).

II. *Arterial duplication.* This abnormality in 44 cases was found on the ACoA — partially in 21 and totally in 23 cases (Fig. 3). Partial duplication indicated that the ACoA had the shape of transversely oriented letter "V" or "Y"; their common origin was more frequent on the left ACA (n = 14) than on the right one (n = 7). Duplicated ACoAs were of different caliber in 21 cases (a caudal vessel was smaller in 19 cases and larger in two cases) and of the same caliber in two specimens. It should be noted that one of two ACoAs was hypoplastic in 23 cases; both ACoAs were hypoplastic in one specimen (case 51). Simultaneously, the left and right A1 parts were of equal outer diameters in 30 cases; the left A1 was larger in eight and smaller in six cases than the right one. Insular atherosclerotic plaques of duplicated ACoAs were found in seven cases; an aneurysm on the rostral ACoA was found twice (cases 19 and 83).

III. *Arterial triplication.* This abnormality in five cases was found on the ACoA; two specimens had independent vessels, whereas three specimens had an association of partially duplicated and single ACoA (Fig. 4). Thereby, one ACoA was hypoplastic in three cases; two ACoAs were hypoplastic in two cases (cases 55 and 58). Simultaneosly, the left A1 was dominant in one case (case 55), as well as the right A1 (case 42).

IV. *Anterior communicating network* or t*he rete communicans anterior.* This ACoA configuration was found in 22 cases (see Fig. 4). The position of anastomotic vessels indicated that the ACoA had the shape of transversely oriented letter "H" or "N" or irregular form. The left and right A1 segments were equal in 16 cases; the left was smaller in five cases and larger in one case than the right A1. Atherosclerotic plaques were found in three cases. An aneurysm of the ACoA existed in only one case (case 57; see Fig. 4).

V. *Arterial quadriplication.* This abnormality was found on the ACoA in two cases. There was an infundibular widening of ACAs at ACoA junction, whereas the outer diameters of the ACAs proximally and distally to it were relatively equal (Fig. 5).

VI. *Transverse arterial anastomoses*. These anastomoses connected bilaterally to A2 segments in three cases; in two cases there was an anastomosis rostral to the duplicated ACoA (case 10, see Fig. 2) and rostral to the rete communicans anterior (case 84; see Fig. 4), and once as multichaneled anastomoses immediately rostral to the single ACoA (case 14; see Fig. 5).

Fig 1. Drawings of 87 abnormalities of the anterior cerebral–anterior communicating (ACA–ACoA) complex.
Note: The number on each drawing will correspond to the same number on one of the next original pictures.

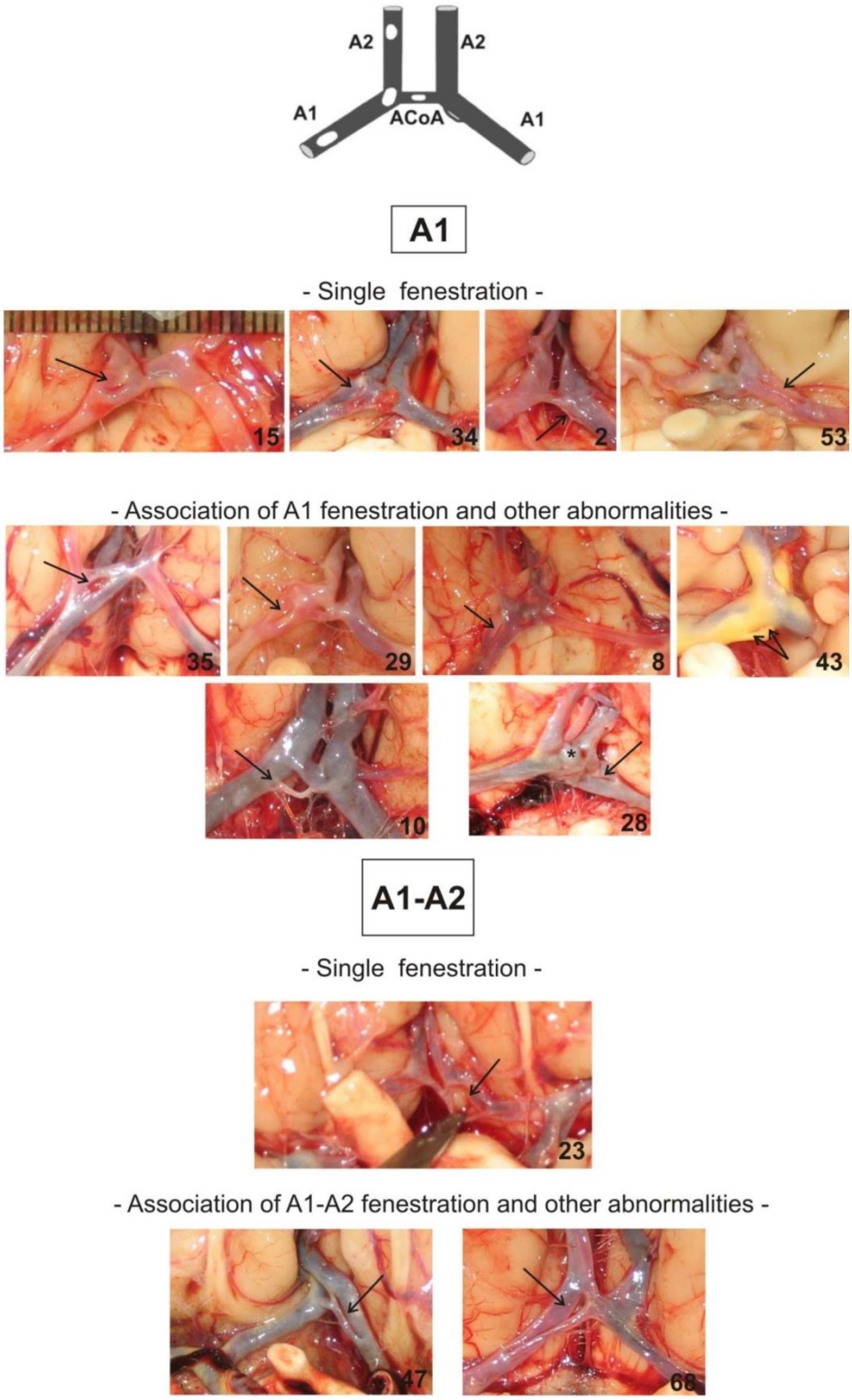

Fig 2. Fenestrations of the left or right anterior cerebral artery (ACA). Unilaterally, fenestration of the precommunicating (A1) and precommunicating–postcommunicating junction (A1–A2) was present in 13 cases. An aneurysm (*) of the anterior communicating artery (ACoA) was present in a case 28.

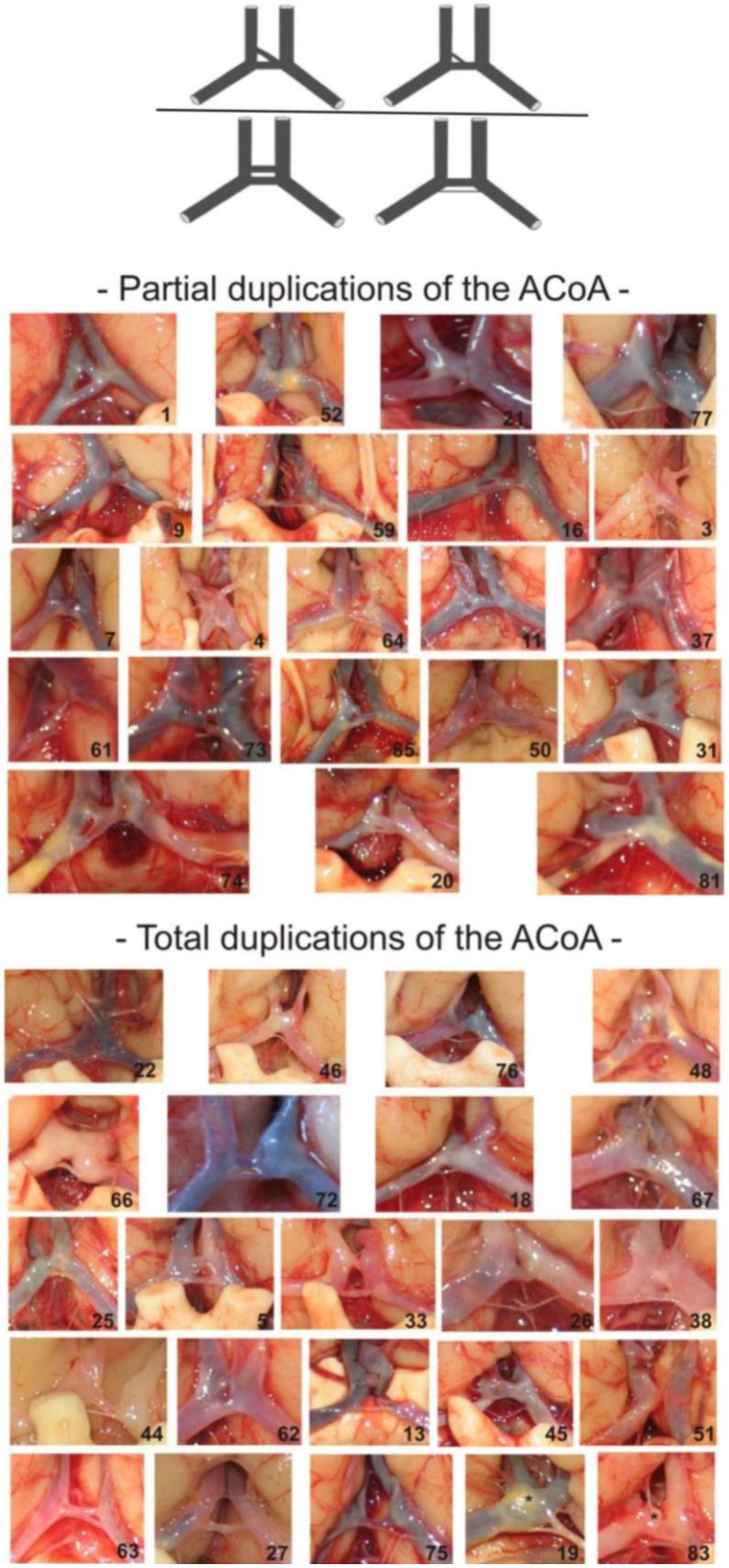

Fig. 3. Duplication of the anterior communicating artery (ACoA). Twenty one specimens characterized by single appearance of partially duplicated ACoA (a common origin on one anterior cerebral artery (ACA) and two origins on the second ACA; 23 specimens characterized by totally duplicated ACoA. An aneurysm (*) of the ACoA was present in cases 19 and 83.

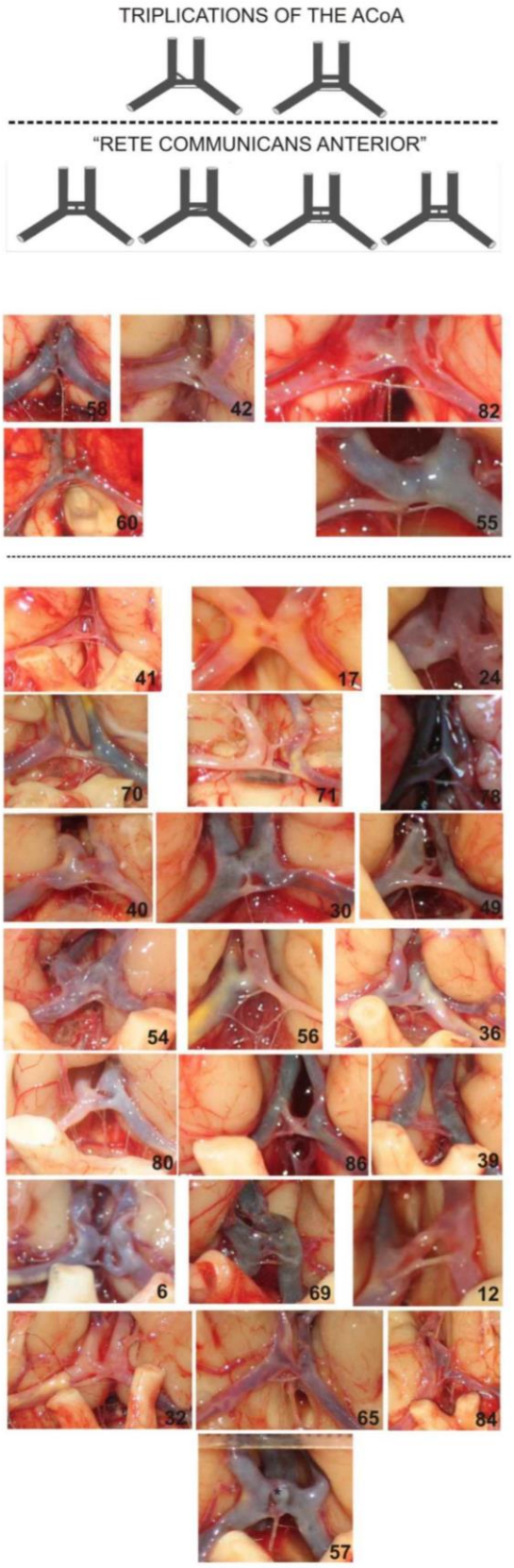

Fig. 4. Triplication of the anterior communicating artery (ACoA) and the rete communicans anterior.Five specimens of ACoA triplication and 22 specimens of the rete communicans anterior. Associated ACoA aneurysm (*) was marked in case 54.

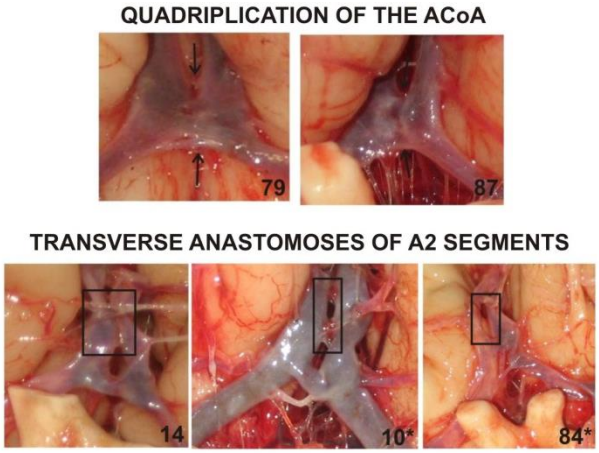

Fig. 5. Very rare abnormalities of the anterior cerebral–anterior communicating (ACA–ACoA) complex: two cases of quadriplication of the ACoA and three cases of multiple transverse anastomoses of postcommunicating parts (A2) of the ACAs; repeated instances from one of the previous images are marked with an asterisk number.

Table 1. Incidences of investigated morphological abnormalities and associated aneurysms of the anterior cerebral–anterior communicating arteries

Arterial abnormalities		A1	A1–A2	A2	ACoA	Percentage in regard to the total number of cases (n = 266)	Percentage in regard to the number of abnormalities
		n					
		Left/Right	Left/Right	Left/Right			
Fenestration	Single	3/6	2/1			4.88%	14.94%
	Double	0/1					(13/87)
	Aneurysm				1	0.37%	7.69% (1/13)
Duplication	Partial				23 (21+2*)	8.64%	
	Total				25 (23+2*)	9.39% 18.04%	55.17% (48/87)
	Aneurysm				2	0.75%	4.16% (2/48)
Triplication	Partial and total				5	1.88%	5.74% (5/87)
	Aneurysm						
Quadriplication	Partial and total				2	0.75%	2.29% (2/87)
	Aneurysm						
Rete	Summa				24 (22+2*)	9.02%	27.58% (24/87)
	Aneurysm				1	0.37%	4.16% (1/24)
Transverse anastomoses	Summa			3 (1+1* +1**)		1.12%	3.44% (3/87)
	Aneurysm						

A1, precommunicating part of the anterior cerebral artery; A2, postcommunicating part of the anterior cerebral artery; A1–A2, junction of the A1 and A2 segments; ACoA, anterior communicating artery.
*One or two cases more in the corresponding square derived from the group of fenestration of the anterior cerebral artery.
**One case more in the corresponding square derived from the group of the rete communicans anterior.

The incidences of investigated abnormalities of the ACA–ACoA complex were summarized in the Table 1. According to the number of specimens and corresponding abnormalities, the incidences were relatively high for ACoA duplications (18.04% and 55.17%, respectively), the rete communicans anterior (9.02% and 27.58%, respectively) and fenestration of the ACA (4.88% and 14.94%, respectively).

However, we found only four aneurysms in specimens of female gender; each was unruptured and lo-

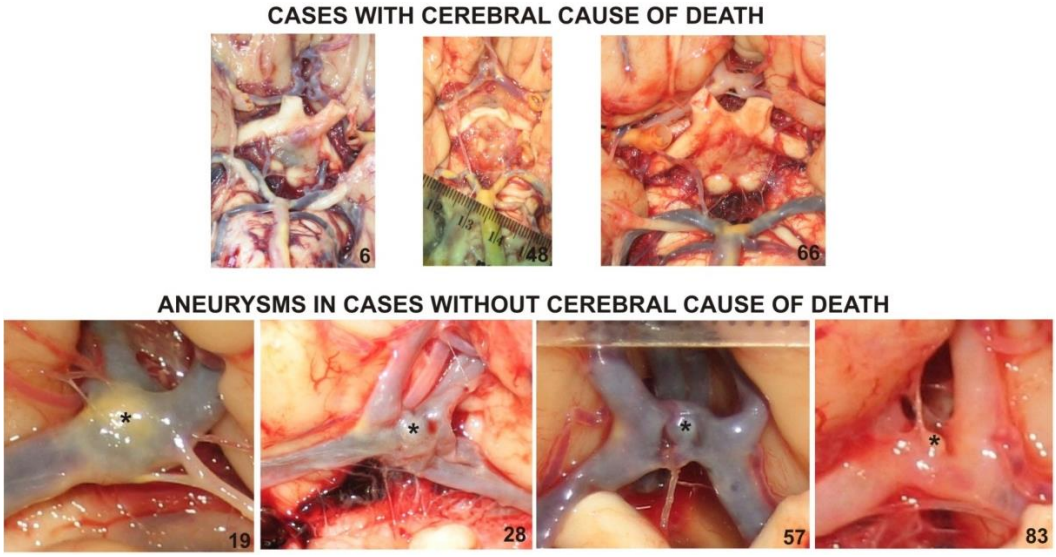

Fig. 6. Comparison of abnormalities of the anterior cerebral–anterior communicating (ACA–ACoA) complex. Three cases were autopsied because of cerebral pathology; ACoA was in the form of the rete communicans anterior (case 6) or doubled (cases 48 and 66). Four cases were without cerebral cause of death, but with a presence of the ACoA aneurysm; ACoA was also in the form of the rete communicans anterior (case 57) or doubled (cases 19, 28 and 83).

Table 2. Display of cadaveric cases with initial cerebral cause of death and cases with proven aneurysms at autopsy

Groups	N^0	Diagnose	Gender/Age	Abnormalities			
				ACoA	ACA		
					L	R	L/R outer diameter
Initial cerebral cause	6*	Cerebral infarction	M/82	Rete**			L=R
	48*	Cerebral/myocardial infarctions	M/77	Duplication		Stenosed origin	L>R
	66*	Cerebral apoplexy	M/68	Duplication			L=R
Anerysmatic serie at autopsy	19*	Massive pneumonia	F/85	Duplication Aneurysm			L<<R
	28*	Polytrauma	F/72	Duplication MACC Aneurysm	A1 fen		L=R
	57*	Respiratory insufficiency	F/95	Rete** MACC Aneurysm			L=R
	83*	Cardiorespiratory arrest	F/74	Duplication Aneurysm			L<R

* The numbers are the same as in Figures 1–4 and Figure 6
** The rete communicans anterior
ACoA, anterior communicating artery; ACA, anterior cerebral artery; M, male; F, female; L, left; R, right; MACC, median artery of the corpus callosum; A1, precommunicating part of the ACA

cated on the ACoA; its incidence was 1.50% (4/266) and 4.59% (4/87) of the cases. We emphasized that cerebral pathology (cerebral infarction or apoplexy) was the reason for the forensic autopsy in 3/87 abnormalities in which aneurysms of the ACA or ACoA did not exist (Fig. 6 and Table 2).

Discussion

As quoted by Okahara et al. [7] a fenestration or duplication of the ACA and/or ACoA is the consequence of an incomplete fusion of the embryonic plexiform anastomosis. As quoted by Koh et al. [24] fenestration of the ACA is a remnant of an embryologic anastomosis between the primitive olfactory artery and the ACA.

Some authors [7,25] described embryonic development of the ACA–ACoA complex as follows: In a 3.7 mm of embryo the primitive internal carotid artery (ICA) gives a large primitive maxillary artery and then divides into the cranial and caudal branches. The cranial branch passes dorsal to the optic stalk, and its first branch is the anterior choroidal artery. It also distributed several branches to the telencephalon, one of which will later form the middle cerebral artery.

The continuation of the cranial branch is known as the primitive olfactory artery [32]. In embryos of 11.5 to 18 mm (41–48 days) the primitive olfactory artery distributed two branches— one to the nasal fossa and the second one as the future continuation of the ACA. The latter artery is joined with opposite ACA by a plexiform anastomosis, which is a precursor of the future ACoA.

We described adult cases of some morphological arterial abnormalities of the A1, proximal A2 and ACoA as parts of ACA–ACoA complex in accordance with Pai et al. [5]. In the literature there were similar examples. So Perlmutter and Rhoton [4] investigated ACA, ACoA and Heubner's artery within the anterior cerebral-anterior communicating-recurrent artery complex. Bharatha et al. [23] included A1, communicating and proximal A2 segment as parts of an anterior communicating region.

Generally, the frequency of fenestrations for all location in the human cerebral circulation was 16% reported at the time of autopsy or 12% angiographically [33]. According to the rule in defining an arterial fenestration accepted in the literature [10,23,25,30,31], we could note the fenestration of the ACA but not ACoA. We found ACA fenestrations in 4.88% of cases, while Bharatha et al. [23] discovered fenestrations on CTAs of the anterior communicating region (A1, ACoA and proximal A2) in 6.9% of cases. Okahara et al. [7] described that their incidence ranged 0.1–7.2% in autopsies. Koh et al. [24] counted 60 cases of A1 fenestration in the literature up to year 2008.

Kim et al. [21] reported that 0.36% of patients who underwent cerebral angiography showed fenestration in A1, while 0.62% of MRA patients had a fenestration of A1 segment. As quoted by Uchino et al. [22], and Dimmick and Faulder [25], very low incidence (0.058%) of ACA fenestrations was noted on angiogram and that was lower than MRA incidence (1.2%). The reason for this was the fact that two fenestrated branches are usually divided horizontally and that they are superimposed on conventional angiographic images [22]. However, Ješić et al. [29] retrospectively reviewed cranial MR angiography images of 1000 consecutive patients and noted 0.5% of ACA fenestrations. In addition Okahara et al. [7] and Weil et al. [34] pointed to the fact that a small fenestration of ACA or partially occluded A1 segment duplication might be mistaken for ACoA aneurysm on MRA and CTA, respectively.

We found only one unruptured aneurysm of the ACoA in the group of 13 ACA fenestrations. This finding can prove the fact that aneurysms rarely develop from ACA fenestrations; untill 1988 only eight cases have been reported in the literature [16]. However, in series of 38 A1 aneurysms, Suzuki et al. [17] discovered vascular anomalies in 20.5% of the cases; 75% of these were A1 fenestrations. In addition, vascular anomalies associated with A1 fenestration were different as an ipsilateral middle cerebral artery aneurysm, an ipsilateral posterior communicating artery aneurysm, and an aneurysm at the proximal end of the fenestrated A1 [21]. Kobayashi et al. [15] reported a case of a ruptured aneurysm occurring at the bifurcation of the azygos ACA with fenestration at the right A1 segment. Ihara et al. [20] discovered an aneurysm from the fenestrated right A1 simultaneously with the presence of the azygos ACA and hypoplastic left A1 segment. Dimmick and Faulder [25] demonstrated on CTA an association of the right A2 and ACoA fenestrations with an aneurysm on the ACoA, then the presence of hypoplastic left A1 and the right persistent primitive hypoglossal artery. Kachara et al. [19] reported a rare case of fenestration of the right Al segment with a ruptured saccular aneurysm, arising from the proximal end of fenestration, associated with aplasia of the left Al segment. Bharatha et al. [23] described that aneurysms associated with fenestrations classically rose at the proximal end supported by hemodynamic stresses and by medial defect at this point. However, Koh et al. [26] reported a case of a ruptured aneurysm arising from the distal end of A1 fenestration. Some authors [21,23,33] stressed that the form of the fenestrations at both the proximal and distal edges is in response to hemodynamic forces and is analogous to branching regions of cerebral arteries. Koh et al. [24] reported an unusual case of multiple fenestrations of the right A1 segment associated with aneurysm of the left A2 segment. Independent from previous data, we agree with the findings of Kayembe et al. [14], Bharatha et al. [23] and Van Rooij et al. [27] that there was no significant difference in the rate of aneurysms in individuals with and without fenestrations, or vice-versa.

Uchino et al. [22] diagnosed that all A1 fenestrations were large, of a convex-lens-like shape, and located in the distal A1 segment with or without extension to the proximal A2 segment, whereas A2 fenestrations were small and of a slit-like shape on MRAs of Japanese population. We found 4/13 large ACA fenestrations in cadaveric specimens of our population.

We reported ACA fenestrations only unilaterally, as recently in adult cadavers as earlier in fetal ones [9, 12]. However, Friedlander and Ogilvy [18] and Aktüre et al. [30] discovered fenestration on both ACAs in two women using 3-dimensional computerized tomographic angiography (3D CTA) and autopsy, respectively. Previous authors summarized only seven cases, including their findings of bilateral A1 fenestrations between 1928 and 2011. In retrospective study by Kim et al. [21], as well as in our specimens there was no side-difference of A1 fenestration, whereas Zhao et al. [28] found it frequently on the right side. There was a dominance of female sex in our and many other reports [10,13,15,16,19, 20,22,24–26,29,30] regarding A1 fenestrations.

In evaluating cerebral angiograms, it is important to differentiate between A1 fenestration and Heubner's artery [12,16]. Perforating arterioles from the medial limb of the ACA fenestration were visible in 12/13 our cases, while Aktüre et al. [30] discovered them from both lateral and medial limbs on postmortem specimen.

Gurdal et al. [19] presented an association of the right A1 fenestration and partial duplication of the ACoA in a female cadaver. We also noted associated morphological abnormalities on the ACoA in six cases, as well an aplasia of the ACoA and opposite ACA and the persistence of the primitive olfactory artery in single cases simultaneously with ACA fenestration.

We did not find any case of ACA doubling, whereas Fawcett and Blachford [6] noted 2/700 cases of "ACA doubling on the right side within the circle". Perlmutter and Rhoton [4] noted duplication of a portion of A1 on one side in 2/50 brains.

It has been reported that the incidence of fenestration of the ACoA was 7.5–40% in autopsies [7]. However, according to the definition of the fenestration, we did not classify any case as an ACoA fenestration. Scremin [8] described that variations of the ACoA include absence, duplication, and triplication. De Silva et al. [11] classified variations of the ACoA into 12 types: single, one point fusion, long fusion, double, V shape, Y shape, H shape, N shape, triple, plexiform, presence of median anterior cerebral artery, and aneurysms. We classified investigated abnormalities of the ACoA into four groups—duplication, triplication, quadriplication and anterior communicating network. We named ACoA in the form of a network as the rete communicans anterior, while Fawcett and Blachford [6] marked similar ACoA configuration as an anterior communicating treble.

It was difficult to discuss incidences of ACoA abnormalities because the same configuration has been presented differently by different authors. This example was a review of findings of different authors or some mistakes in explanation of other results as there were in the table presented by De Silva et al. [11]. We found the anterior communicating network and total duplication of the ACoA in 9.02% and 9.39% of cases, respectively, while Fawcett and Blachford [6] noted them in legends of their drawings in 14% and 7.2% of cases, respectively. We defined a triplication of the ACoA if there were three independent vessels or an association of partially duplicated and single ACoA, whereas Gurdal et al. [10] presented a partial triplication of the ACoA when it had a common orifice on one ACA and trifurcation on opposite ACA.

There are some our suggestions. The first is the consequence of the summation of double to multiple ACoAs and anterior communicating network (21.32% and 9.02%, respectively). We suggest including the plural for ACoAs and a new term—rete communicans anterior in Anatomical nomenclature. Another suggestion was the consequence of an indefinite distance between two or more successive ACoAs and an unspecified border for the most rostral ACoA. Because of that we included a group of transverse ACAs anastomoses as a supplementary abnormality of the ACA–ACoA complex.

Finally, we mentioned in this manuscript only one case (1/266) of absence of the left ACA and ACoA in specimens of Serbian population. However Uchino et al. [22] found 50/923 instances of unilateral A1 aplasia in Japanese population. This important abnormality, as well as possible cases of an "induced" ACoA aplasia deserves special consideration in the future article.

Conclusion

We found morphological abnormalities of the ACA–ACoA complex in 87/266 or 32.71% of cases. The finding of only four aneurysms on the ACoA indicates that there was no significant difference in the rate of aneurysms in individuals with and without fenestrations or duplications or multiplications of the ACA–ACoA complex. This fact inspired authors for additional investigation of relationships of abnormalities with or without aneurysms on other arteries of carotid and vertebrobasilar systems in our population.

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