Importance of the microsurgical anatomy of the A1-anterior communicating artery complex

Alejandro Monroy-Sos,¹ Julio César Pérez-Cruz,¹ Gervith Reyes-Soto,² Carlos Delgado-Hernández,¹ Mario Alberto Macías-Duvignau,¹ Luis Delgado-Reyes²

Abstract

Background: The anterior cerebral artery originates from the bifurcation of the internal carotid artery lateral to the optic chiasm, joining with its contralateral counterpart via the anterior communicating artery (ACoA). The A1-ACoA complex is the most frequent anatomic variant and is the major site of aneurysms in between 30 and 37% of patients. We carried out this study to determine the microsurgical anatomy, anatomic variants and importance of complex precommunicating segment-ACoA in neurological surgery of vascular pathology, mainly aneurysms, in a Mexican population.

Methods: The study was performed in 30 injected brains. Microanatomy was studied (length and diameter) of A1-ACoA complex and its variants.

Results: There were 60 A1 segments. Average length of the left side was 11.35 mm and for the right side was 11.84 mm. The average diameter of the left side was 1.67 mm and the right side was 1.64 mm. The average number of perforations on the left side was 7.9 and the right side was 7.5.

The ACoA was found in 29 brains of the optic chiasm. Its course depended on the length of the A1 segment. The average length of the segment was 2.84 mm, the average diameter was 1.41 mm and the average number of perforations was 3.27.

The A1-ACoA complex variants were found in 18 (60%) along with the presence of two blister-like aneurysms.

Conclusion: It is necessary to understand the microanatomy of the A1-ACoA complex and of its variants in order to have a three-dimensional vision during aneurysm repair surgery.

Key words: anterior cerebral artery, anterior communicating artery, anatomic variants, blister-like aneurysm.

Introduction

The anterior cerebral artery arises from the bifurcation of the internal carotid artery, lateral to the optic chiasm, in front of the anterior perforated substance (APS). It then enters into the interhemispheric fissure where it joins its contralateral homologue via the anterior communicating artery (ACoA).

The precommunicating artery-anterior communicating segment is the location with the most anatomic variants: hypoplasia, aplasia, duplicate, triplicate, formed in V, Y, H, N, plexiform disposition, fenestrations, trabecular indentations, multicannulated and infraoptic course. The ACoA is the most frequent site (30-37%) for intracranial aneurysms. These are the most complex and difficult to manage in their anterior circulation because of the angioarchitecture, dynamic circulation flow in the region of the ACoA and frequent anatomic variants that are occasionally not well known and are not visible on preoperative imaging.

Cerebrovascular disease (CVD) is the sixth leading cause of death in Mexico. Between 5 and 10% correspond to subarachnoid hemorrhage; the majority of cases occur due to the rupture of an aneurysm with fatal hemorrhage in 50%.

The purpose of the study is to determine the microsurgical anatomy, anatomic variants and the importance of the segment of the precommunicating artery-ACoA complex in...
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Materials and Methods

We carried out a prospective and descriptive study in the Department of Anatomy, School of Medicine, National Autonomous University of Mexico (UNAM). Thirty brains were obtained from a Mexican population between 20 and 55 years of age, and 60 hemispheres were, respectively, analyzed. Excluded from the study were brains with a history of trauma. For all brains, a washing of the arterial system was carried out with saline solution 0.9% for 45 min after which they were injected with red latex and formaldehyde 10% and remained as such for 3 weeks. Microsurgical dissection of the precommunicating artery-anterior communicating segment was performed with a ZEISS-OPMI-1 microscope low magnification with 5X and 10X. For measurements, an electronic calibrator UPM model 111-523 with a resolution of 0.01 mm and accuracy of ±0.02 mm was used. The diameter and the length of the precommunicating segment and ACoA were measured and the number of protruding perforants and morphometric description of the anatomic variants found. The hypoplastic segments were considered when the caliber was <1 mm and asymmetry if the diameter of one of the segments corresponded to 75% or less of the contralateral segment.

Results

Anterior Cerebral Artery: Precommunicating Segment A1

The precommunicating segment or first segment of the anterior cerebral artery (A1) had an anteromedial course over the optic chiasm. Occasionally it was tortuous due to the short or asymmetric length of the precommunicating segment of its contralateral segment. It has been reported that it courses over the optic or infraoptic nerve; however, in our study it did not appear in this manner. It subsequently united with the contralateral segment by means of the ACoA to continue as a postcommunicating segment in the interhemispheric fissure.

There were 60 segments found in the first segment of the anterior cerebral artery. The average length of the left precommunicating segment was 11.35 mm (range: 8.7-14.5 mm) and the right was 11.84 mm (range: 8.4-16.08 mm). The average diameter of the left precommunicating segment measured 1.67 mm (range: 1.07-2.17 mm), and the right was 1.64 mm (range: 0.3-2.3 mm). The average number of perforants of the left precommunicating segment was 7.9 (range 4-12) and the right was 7.5 (range: 3 to 13) (Table 1). The perforating arteries are divided into two groups: medial lenticulostriated and recurrent artery of Heubner (RAH). The greater part of the perforating arteries originated from the posterolateral and proximal portion of the A1 segment, of which about half were directed towards the APS and, subsequently, to the globus pallidus, caudate nucleus and left anterior arm of the internal capsule. The remaining were distributed to the dorsal portion of the optic chiasm, optical tract, optic nerve, mid-third of the anterior commissure, suprachiasmatic portion of the hypothalamus and interhemispheric fissure. When the perforating arteries originated from the distal precommunicating segment they were directed, in their majority, towards the optic chiasm. Bilateral origin was found in the RAH in the lateral and distal portion of the precommunicating segment in nine brains (30%) (Table 1).

Anterior Communicating Artery

The ACoA, or vascular bridge, is the union between the two precommunicating segments. It was found in 29 brains over the optic chiasm. Its trajectory depended on the length and the diameter of the precommunicating segment. It was occasionally seen in a horizontal, oblique or tortuous direction. In the study the largest number was found in the oblique direction. The average length of the ACoA segment was 2.84 mm (range: 0.4-4.28 mm) and average caliber of 1.41 mm (range: 0.32-2.3 mm). The average number of perforants was 3.275 (range: 2-5) (Table 1).

The perforating arteries came out of the medial portion of the communicating segment and its posterior face in an isolated manner or through the principal trunk. The perforants coursed towards the optic chiasm, lamina terminalis, optic nerve and anterior portion of the hypothalamus. The origin of the RAH was found in five brains (16.6%) adjacent to the ACoA segment.

Variants of the Precommunicating Segment-Anterior Communicating Artery (Table 2)

In 18 brains (60%) we found variants of the precommunicating segment-anterior communicating artery A1-ACoA. The most common was hypoplasia of the anterior communicating artery, which was found in five brains (16.6%). In the precommunicating segment A1, the hypoplastic variant was found in one brain (3.3%).

A duplicated ACoA was found in four brains (13.3%). In two of these there was a “very hypoplastic” segment, the expression used by Yasargil to describe hypoplasias <.5
mm. In another brain the hypoplastic variant was found in V and Y forms (Figure 1) and in the fourth brain the ACoA segment was associated with a trabecular indentation. The fenestration variant was found in three brains: two in the anterior communicating segment (6.6%) (Figure 2) and one (3.3%) in the distal part of the precommunicating A1 segment. The multicannulated variant was found in one brain (3.3%) and, finally, the ACoA was found in triplicate in 3.3% (Figure 3). Two blister-like aneurysms were found: the first in the communicating segment with anterior projection associated with a duplicate variant, hypoplastic and with a subcallosal artery (Figure 4).

The second blister-like aneurysm with anterior projection was associated with a duplicate and hypoplastic variant and a common trunk or frontobasal that gave rise to the RAH, orbitofrontal and frontopolar artery (Figure 5).

In one brain (3.3%) an artery azygos was found, formed by the asymmetrical union of the precommunicating A1 segments with a right-sided predominance with an odd number of the postcommunicating A2 segment. The right side of the precommunicating distal A1 segment showed a common trunk with origin of the orbitofrontal, frontopolar artery, and the RAH. On the left side, the distal precommunicating A1 segment showed the RAH and orbitofrontal artery. This variant was found to be associated with aneurysms; however, a meningioma was observed in the right temporopolar face (histopathological study) (Figure 6). The subcallosal artery is a long branch that originates from the midportion of the ACoA. It was found in two brains (6.6%) with a dorsocaudal path to the interhemispheric fissure, its perforating branches directed toward the optic chiasm and part of the anterior hypothalamus (Figure 4). The RAH has a mean of 23.2 mm on the right hemisphere and on the left of 22.8 mm. The origin of the RAH is asymmetrical in the anterior cerebral artery segments because it is only symmetrical bilaterally in nine precommunicating A1 segments, five in the communicating segment and 12 in the postcommunicating segment. The RAH was found to be asymmetric in three brains and absent in one hemisphere. It was found to be duplicated in one brain and its origin in two brains was through the common trunk or frontobasal artery. The average of the perforating branches was found to be between 3 and 6.

### Discussion

The precommunicating A1 segment varies in length with an average of 12.7 mm and a caliber of 2.37 ± 0.68 mm. In our series a length and diameter were reported that agree with the literature. Hypoplasia of the precommunicating A1 segment was found by Yasargil in 80%, Karasincir et al. in 26.6%, Suzuki in 68.1%, Rhoton in 10%. Tao et al. did not find a hypoplasia <1 mm; however, they introduced the term “disproportionate development,” which was found with a frequency of 13.30% in our study we found hypoplasia of the precommunicating segment in 3.3%. Rhoton

### Table 1. Measurement of diameter, width, and number of perforants of the A1–ACoA complex

<table>
<thead>
<tr>
<th></th>
<th>A1 left</th>
<th>A1 right</th>
<th>ACoA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Length</td>
<td>A: 11.35 mm SD: 1.81</td>
<td>A: 11.84 mm SD: 2.216</td>
<td>A: 2.84 mm SD: 1.366</td>
</tr>
<tr>
<td>Diameter</td>
<td>A: 1.67 mm SD: 0.46</td>
<td>A: 1.64 mm SD: 0.467</td>
<td>A: 1.41 mm SD: 0.560</td>
</tr>
<tr>
<td>Perforants</td>
<td>A: 7.9 SD: 2.309</td>
<td>A: 7.5 SD: 2.569</td>
<td>A: 3.275 SD: 0.751</td>
</tr>
</tbody>
</table>

A1, precommunicating segment; ACoA, anterior communicating artery; A, average; SD, standard deviation.

### Table 2. Variants of A1-ACoA segment and aneurysms

<table>
<thead>
<tr>
<th>Variants</th>
<th>A1 left</th>
<th>A1 left</th>
<th>ACoA</th>
<th>A2 Impar</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypoplasia</td>
<td>1</td>
<td></td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>Duplicate</td>
<td></td>
<td>4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Triplicate</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fenestration</td>
<td>1</td>
<td>2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Multicannulated</td>
<td></td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Subcallosal artery</td>
<td>2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Azygos artery</td>
<td></td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aneurysms</td>
<td>2</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

A1, precommunicating segment; ACoA, anterior communicating artery; A2, postcommunicating segment.
reported that hypoplasia of the precommunicating segment is associated with aneurysms in 85%. Suzuki and Tao et al. reported only one case of aneurysm in their series. We found no aneurysm in the precommunicating segment with hypoplasia. Rhoton, Stehbens and Suazo et al. report that hypoplasia of the precommunicating segment is the only anatomic variant that can be associated with aneurysm of the precommunicating artery-ACoA complex. The asymmetries in the complex are of great importance in the
genesis of aneurysms as the impact of the flow of the dominant precommunicating segment against the arterial wall is the factor that triggers the formation of aneurysms.9,22 We found in our study asymmetry of the precommunicating A1 segments in 76.6% without an aneurysm relationship.

Aneurysms of the precommunicating A1 segment are rare and are commonly associated with other vascular abnormalities such as plexiform disposition or fenestrations in the precommunicating A1 segment, accessory middle cerebral artery, tortuous precommunicating A1 segment and azygos artery.23,24 Fenestrations have been found in between 0 and 4% and are often associated with aneurysms.25 This study found variants of the precommunicating A1 segment without a relationship to aneurysms.

The RAH may originate in the precommunicating A1 segment, ACoA or in the postcommunicating A2 segment. The RAH is frequently directed towards the anterior third of the putamen, anterior segment of the caudate nucleus, anterior segment of the globus pallidus, anterior branch of the internal capsule and the uncinate fasciculus.26 The course of the RAH must be identified so as to prevent damage during a definitive temporal clipping or when resecting the straight gyrus for management of complex aneurysm of the ACoA. Gomes et al.27 described three different paths of the RAH in relation with the A1 segment: a superior path (64%), anterior path (34%) and posterior path (3%).

The azygos artery was described by Wilder in 1885 with the name “arteria termatica” and was described as a fusion of both postcommunicating A2 segments. There are two theories for the formation of the azygos artery: one is the abnormal fusion of the A2 segments from the medial branch of the primitive olfactory artery and the second is the persistence of the middle artery of the corpus callosum.10,28-30 The international literature reports it at 0.0-5%. Yasargil17 found it in 2% of anatomic dissections. Huber et al.30 carried out angiographic studies and reported that there may be a relationship with aneurysm formation in 41.1%. The anterior azygos cerebral artery is associated with various malformations: agenesis of the corpus callosum, hydroanencephaly, distal saccular aneurysms with a frequency between 13 and 71%, nonsaccular in 0.5% and vascular malformations.31-33 In our series no aneurysms of the azygos variant were found.

The ACoA originates in the embryo as a multicannulated vascular network that subsequently evolves to a single vessel. This may be the cause of the great anatomic variety and of the possibility of aneurysms due to histological and hemodynamic changes.28 Yasargil et al.3 and Marinkovic10 in their studies grouped the anterior communicating segment into simple and complex based on its morphology. The simple type refers to only one bridge and the complex has two branches in X, Y, O, H or reticular shape. Tao et al.19 describe 31.10% as simple, 64.4% complex and 4.40% with fused precommunicating A1 segments. Our study found 76.6% as simple type and 23.3% complex type and 3.33% with fused precommunicating A1 segments without ACoA.

Duplication of the ACoA is important in cases in which the aneurysm has a dome towards the front and the arterial bridge that causes the variant could be confused with the neck of the aneurysm and be accidentally clipped.34 The incidence of ACoA duplication is from 5.7-11% with aneurysm.35 In our study, aneurysms were found in 6.6% with a duplicated variant. The microsurgical anatomy of the perforating arteries of the complicating segment is complicated and there is still controversy.36 The number of perforants varies: <3, not greater than 6, and between 1 and 11.37 In our study we found the perforating arteries were between 2 and 5, which coincides with the international literature. The perforants that are found on the base of the aneurysm of the ACoA could be damaged during dissection, coagulation or clip application. This is particularly true for aneurysms with a wide base and anatomic variants.39,40

The relationship between anatomic variants of the precommunicating complex-ACoA and saccular and nonsaccular aneurysms has been frequently reported in the literature, but for blister-like aneurysms there have only been five surgical cases reported and none during anatomic dissection. Blister-like aneurysms are described as a small hemispheric protuberance with clinical characteristics different from the saccular aneurysms. The blister-like aneurysms occur...
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most frequently in the dorsomedial wall of the supraclinoid segment of the internal carotid artery. There are various causes that give rise to them; those that appear to be the most important are hemodynamic stress and atherosclerosis. Andaluz and Zuccarello report that in three cases the blister-like aneurysms were not detected with digital subtraction angiography, which could be explained by the variants of the precommunicating-ACoA complex. Treatment for blister-like aneurysms is difficult due to their wide base and friable walls. The main treatment option is clipping and, occasionally, to wrap the aneurysm or to perform a bypass. These aneurysms are not amenable to endovascular treatment. These rare lesions should be considered when there is subarachnoid hemorrhage of unknown cause or they should be looked for using angiotomography.

There are reports of saccular aneurysms with hypoplasia of the precommunicating A1 segment. In our series we reported two blister aneurysms unrelated to the precommunicating A1 segment hypoplasia or with the duplicated variant of the ACoA. It is necessary to know the location of the microsurgical anatomy of the five vessels (left-right precommunicating A1 segment, ACoA and left right A2) and perforants, specifically the RAH that could be damaged during dissection, resection of the gyrus rectus or clipping. It is necessary to carry out anatomic studies of our population because occasionally what is reported in the literature does not coincide with what is actually found in the experience of each surgeon.

Table 3. Comparison of variants, RAH and aneurysm

<table>
<thead>
<tr>
<th>Hypoplasia</th>
<th>A1</th>
<th>A1 ACoA variants</th>
<th>Origin of the RAH</th>
<th>A1 ACoA A2</th>
<th>A1 ACoA A2</th>
<th>A1 ACoA A2</th>
<th>A1 ACoA A2</th>
<th>Monroy-Delgado</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>80%</td>
<td>10%</td>
<td>42%</td>
<td>1.5%</td>
<td>38%</td>
<td>25%</td>
<td>3.3%</td>
<td>28.3%</td>
</tr>
<tr>
<td>ACoA variants</td>
<td>10%</td>
<td>4%</td>
<td>A1 14%</td>
<td>ACoA 8%</td>
<td>A2 78%</td>
<td>A1 4%</td>
<td>ACoA 58%</td>
<td>ACoA 15%</td>
</tr>
<tr>
<td></td>
<td>42%</td>
<td>64.4%</td>
<td>A1 7%</td>
<td>ACoA 6%</td>
<td>A2 46.0%</td>
<td>A1 4%</td>
<td>ACoA 58%</td>
<td>ACoA 15%</td>
</tr>
<tr>
<td></td>
<td>40%</td>
<td>60%</td>
<td>AC1 6%</td>
<td>ACoA 58%</td>
<td>A2 23%</td>
<td>A2 23%</td>
<td>ACoA 58%</td>
<td>ACoA 15%</td>
</tr>
<tr>
<td></td>
<td>64.4%</td>
<td>50%</td>
<td>A1 28.3%</td>
<td>ACoA 15%</td>
<td>A2 53%</td>
<td>TC 3.3%</td>
<td>ACoA 58%</td>
<td>ACoA 15%</td>
</tr>
<tr>
<td>Azygos artery</td>
<td>2.1%</td>
<td>4.4%</td>
<td>Hypoplasia A1</td>
<td>4.4%</td>
<td>ACoA</td>
<td>ACoA</td>
<td>3.3%</td>
<td>ACoA Duplicate</td>
</tr>
<tr>
<td>Aneurysm</td>
<td>4.4%</td>
<td>ACoA Duplicate</td>
<td>ACoA Duplicate</td>
<td>3.3%</td>
<td>ACoA</td>
<td>ACoA</td>
<td>3.3%</td>
<td>ACoA Duplicate</td>
</tr>
</tbody>
</table>

A1, precommunicating segment; A2, postcommunicating segment; RAH, recurrent artery of Heubner; ACoA, anterior communicating artery; CT, common trunk.

References


