MEDIAN ARTERY OF THE CORPUS CALLOSUM IN THE CONTEXT OF ANTERIOR COMMUNICATING ARTERY ANEURYSM RUPTURE: THE RELEVANCE OF PERIANEURYSMAL ANATOMY

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Abstract

It is important to understand the patient's vascular anatomy before treating cerebral aneurysms. The middle artery of the corpus callosum is one of the least common variations of the anterior communicating artery (AComA) complex. We describe the case of a 59-year-old woman who suffered a subarachnoid hemorrhage due to an AComA complex aneurysm that had ruptured. Fluorescein injection during the aneurysm clipping procedure revealed a partial obstruction of the middle artery, requiring clip repositioning. The vascular variations that patients may exhibit must be considered in aneurysm clipping surgery.

Keywords: *AComA aneurysm, clipping, vascular abnormalities, subarachnoid hemorrhage, fluoresceine.*

Background

The anterior communicating artery (AComA) has been identified around 41-48 days of gestation during embryological development.[1] The AComA develops from a multi-channeled vascular network that coalesces at the time of birth and unites the anterior cerebral arteries (ACA) in the lamina terminalis cistern,[2,3] completing the anterior circle of Willis.[4] In the embryo, it can reach a maximum length of 18 mm.[5] The average AComA diameter is 1.2 mm when the difference in diameter between the right and left precommunicating (A1) segments of the anterior cerebral artery (ACA) is 0.5 mm or less, and 2.5 mm if the difference is more than 0.5 mm.[1,6] A variety of vascular abnormalities of the anterior communicating artery complex include AComA aplasia, duplication, A1 hypoplasia or aplasia, azygos, and median artery of the corpus callosum (MACC).[2,3,4,7,8,9]

The median artery of the corpus callosum is found in 2% of cases and dissections.[2,10,11] It runs parallel to and behind the normal pericallosal artery,[12] and gives branches to the corpus callosum, splenium, and paracentral lobules of both sides.[6,7] Its diameter ranges between 0.4 and 3.1 mm, with an average of 0.9 mm.[9] There are two anatomic variants of this vessel, the classical and hemispheric, the first one terminating along the body of the corpus callosum in the midline and the second one serving as a second pericallosal with medial cortical branches.[9] It is well known that hemodynamic abnormalities create stress in artery bifurcations, resulting in aneurysm formation.[6]

Considering all vascular abnormalities of the AComA complex, we present a case of an AComA aneurysm with a median artery of the corpus callosum.

Case presentation

A 59-year-old woman with a previous history of smoking and alcohol consumption, poorly controlled chronic arterial hypertension, and associated obesity, with a body mass index (BMI) of 40 kg/m2, arrived at the emergency room due to cephalalgic syndrome with secondary characteristics consisting of frontotemporal bilateral intense severity associated with loss of consciousness and posterior recovery. The patient presented transient motor aphasia with full recovery after one hour. Physical examination revealed morbid obesity with no other findings. Neurologic examination showed meningeal signs with a positive jolt accentuation maneuver and nuchal rigidity. Simple computerized tomography (CT) scan showed a subarachnoid hemorrhage and a left Sylvian clot corresponding to Fisher I, Hunt-Hess II, and World Federation of Neurological Surgeons (WFNS) I. CT angiography revealed a superior projecting anterior communicating aneurysm and a median callosal artery (Figure 1).
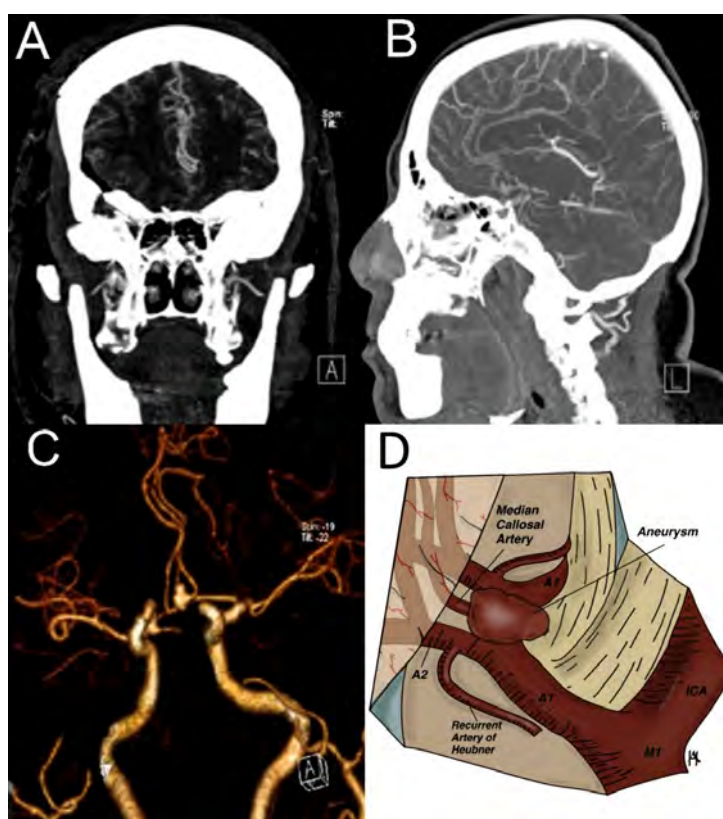

Figure 1. **Preoperative aneurism. A, B**. Preoperative CT angiography in coronal and sagittal projections with evidence of an AComA aneurysm with a superior projecting dome and a median callosal artery. **C**. 3D reconstruction showing the vascular preoperative relations of the aneurysmal dome. **D.** Anatomy diagram of the anterior communicating complex with the aneurysm. CT: computerized tomography; AComA: anterior communicating artery; 3D: three-dimensional; A1: precommunicating segment; A2: infracallosal segment; Ml: sphenoidal segment; ICA: internal carotid artery.

Urgent surgery with standard pterional approach was performed, followed by a sub-frontal corridor and posterior depletion of the carotid and optic-carotid cisterns. Optical nerves, chiasm, carotid artery, and ACA were identified. Opening of the lamina terminalis was performed to achieve brain relaxation. A1 was identified and followed toward the AComA. An anterior-inferior bi-lobulated projecting aneurysm was identified. Dissection of the aneurysm dome was done with bipolar forceps and aspirator. We identified both the infracallosal (A2) arteries; a third artery arising from the AComA was also identified. After the correct identification of all the AComA complex arteries, definitive clipping was performed with a 9 mm straight clip. Fluorescein was administered in a 4 mg/kg dose to confirm parent vessel patency and total aneurysm occlusion. When exposing both A2, a median callosal artery was detected partially occluded by the clip, which made it necessary to relocate it. Aneurysm residual was found in the inferior portion, and a second definitive 5 mm straight clip was used with no residual after clipping. Dural watertight reconstruction, bone repositioning, and aponeurotic repair were performed in the usual manner (Figure 2).

A postoperative CT- angiography (Figure 2) confirmed total occlusion of the aneurysm neck with no residual. The patient evolved satisfactorily with no vasospasm during follow-up and was discharged after six days.
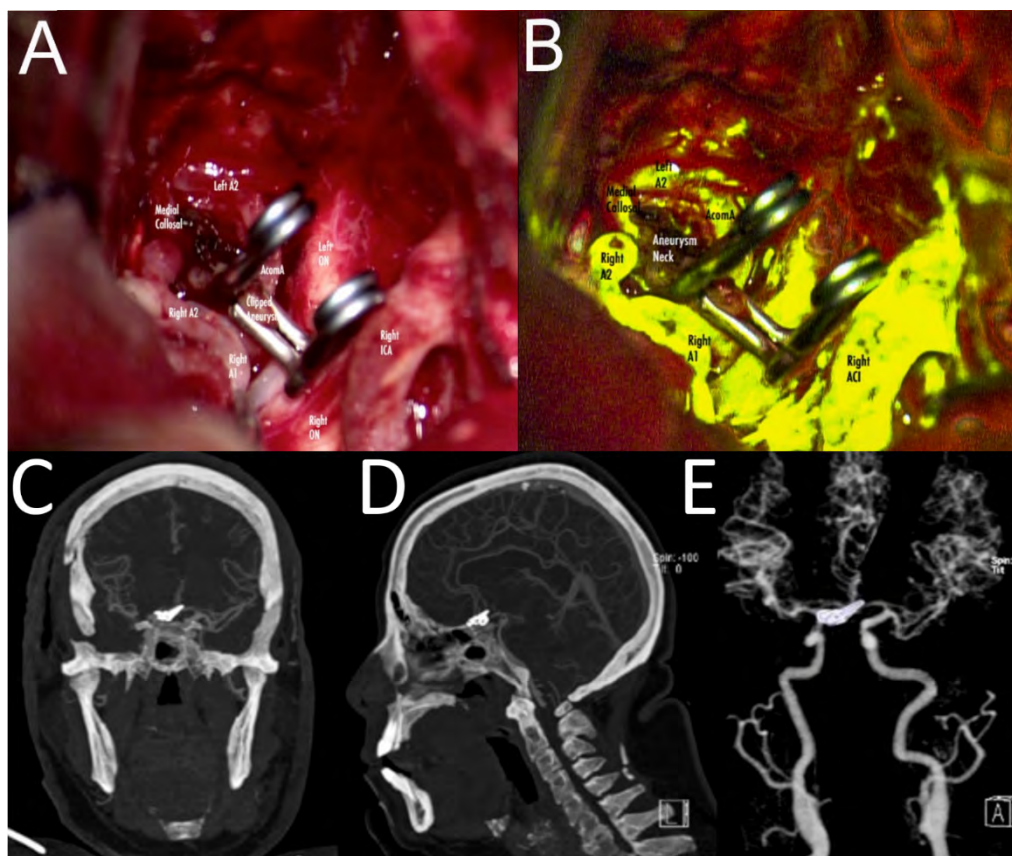

Figure 2. Transoperative photograph and postoperative CT angiography. A. Transoperative photograph showing the AComA complex and the final clip display with no residual. B. Intraoperative fluorescein angiography control with complete patency of the AComA complex and no residual filling of the aneurismal dome. C, D. CT angiography in coronal and sagittal projections displaying clipped AComA aneurysm with a superior projecting dome without residual, and a patent AComA complex with median callosal artery. E. 3D reconstruction of CT angiography showing clip configuration. A1: precommunicating segment; A2: infracallosal segment; M1: sphenoidal segment; ICA: internal carotid artery; ON: optic nerve; AComA: anterior communicating artery. CT: computerized tomography; 3D: three-dimensional.

Discussion and conclusion

Since the exposure obtained in the laboratory is different from that obtained in the operating room, adequate knowledge of the anterior communicating artery complex and its variants is required; this also becomes relevant when considering that the most common aneurysm site on the anterior cerebral artery is at the level of the AComA, which represents the most frequent intracranial aneurysm.[6]

As described by Yasargil and simplified by Lawton, AComA aneurysm dissection identifies 14 arteries: ipsi- and contralateral A1 segments; ipsi-and contralateral A2 segments; AComA; ipsi-and contralateral recurrent arteries of Heubner; ipsi-and contralateral orbitofrontal arteries; ipsi-and contralateral frontopolar arteries, and the collection of AComA hypothalamic perforators, the proximal origin of the callosomarginal arteries and a third A2 segment.[2,3,4]

Multiple surgical approaches have been used to treat anterior communicating artery aneurysms as the operative field is mainly dependent on the selected approach and the amount of dissection made for exposure.

All possible variants must be identified during the preoperative angiography because this will allow the surgeon to select the best approach and operative plan. This also saves time in the operating room since differentiating the median artery of the corpus callosum and an early branching pattern of the A2 may be challenging during surgery. In addition, MACC can be difficult to identify because it runs parallel to and behind the normal pericallosal artery, and it is easy to damage during surgery.[2] As Ogawa et al[13] have mentioned, there are two types of patterns: type A is when the aneurysm is at the trifurcation of the MACC, the branching point of the AComA, and the ipsilateral A1 or A2; type A2 is when the aneurysm is formed at the junction of the AComA and the ipsilateral A.

In this case, previous acknowledgment of the MACC variant allowed extending the AComA dissection until the complete identification of the aneurysm; both A2 and the MACC had a Type A pattern. It is important to highlight that even if a vascular anomaly has not been identified in the preoperative studies, all these variations should be considered, including a MACC, to avoid a postoperative stroke.

Preoperative diagnosis of this anatomic variation is mandatory to avoid occlusion of this artery. Despite not being identified in the preoperative image, it should always be considered that this variation may be present and intentionally discard this possibility to avoid ischemic complications secondary to the occlusion of these abnormal arteries.

Declaration of no interests
The authors declare that they have no conflict of interest to disclose.

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