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Reporte de caso

Accessory middle cerebral artery associated with two intracranial aneurysms

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CEREBRAL MEDIA ACCESORIA ASOCIADA A DOS ANEURISMAS

RESUMEN

Objetivo e importancia: aunque ya publicado, la duplicación de la arteria cerebral media (ACM) es un hallazgo angiográfico reconocido infrecuentemente. En este reporte, la anomalía fue asociada con dos aneurismas, uno roto. *Presentación clínica:* una ACM anómala, tipo gemela, que nace a partir de la arteria cerebral anterior izquierda dominante en segmento precomunicante extendiendose hacia la fisura silviana, en estrecho paralelismo con el segmento esfenoidal de la ACM, fue identificada angiográficamente como una ACM *accesoria*. La paciente fue una mujer de 74 años investigada por un episodio reciente de hemorragía subaracnoidea. Se demostraron dos aneurismas intracraneales: uno originado en la arteria comunicante anterior y el otro en la unión del segmento M1-M2 derecho. *Intervención:* fue rechazado el tratamiento quirúrgico o endovascular, pero la paciente permaneció hospitalizada. Dos semanas después del internamiento, ocurrió un deterioro neurológico súbito asociado a hidrocefalia aguda, tratada con derivación ventricular al exterior de urgencia. Desafortunadamente, la paciente no mejoró, falleciendo como resultado de una neumonía nosocomial. No se otorgó el consentimiento de autopsia. *Conclusión:* se reporta un ejemplo de ACM accesoria, en este caso asociado con dos aneurismas arteriales intracraneales, uno roto. Se hace una breve discusión de la literatura médica.

Palabra clave: ACM accesoria, aneurisma intracraneal, anomalías ACM, aneurismas intracraneales múltiples.

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ABSTRACT

Objective and importance: albeit very likely underdisclosed, duplication of the middle cerebral artery (MCA) is a rather unfrequently recognized angiographic finding. In this instance the anomaly was associated with two intracranial aneurysms, one ruptured. *Clinical presentation:* an anomalous, twin-like MCA, departing from a dominant left pre-communicating anterior cerebral artery and extending recurrently toward the sylvian fissure, in close parallelism with the sphenoidal MCA segment, was angiographically identified as an *accessory* MCA. The patient was a 74 year old woman being investigated for recent spontaneous subarachnoid hemorrhage. In addition, two intracranial arterial aneurysms were also demonstrated: one originated from the left corner of the anterior communicating artery; the other expanded at the right M1-M2 junction. *Intervention:* the proposed surgical or endovascular treatment was refused but the patient remained hospitalized. Sudden neurological deterioration occurred two weeks after hospitalization due to acute hydrocephalus so that an external ventricular drainage system was expeditiously applied. Regretfully, the patient did not improve and expired thereof as result of nosocomial pneumonia. Necropsy request was not granted. *Conclusion:* we hereby report a new example of *accessory* MCA, in this case associated with two

intracranial arterial aneurysms, one ruptured. A brief discussion of the pertinent medical literature is made.

Key words: accessory MCA, intracranial aneurysm, MCA anomalies, multiple intracranial aneurysms.

A list of all anatomical vascular variants of the brain should likely begin with the arterial circle extended at the base of the brain, for a balanced arrangement of its components and proximal branches, a so-called "normal" or "symmetrical" circle of Willis, is observed in not quite 50% of cases[1]. In spite of being the most complex, and have the largest distribution of all intracranial arteries, anomalies of the middle cerebral artery (MCA) are, perhaps paradoxically, less frequently recognized, or reported, than those in other major cerebral vessels. Perusal of the literature reveals several instances of MCA *duplication*, the anomalous channel departing either from the internal carotid artery (ICA) or the anterior cerebral artery (ACA), as well as some examples of proximal MCA *fenestration*.

This paper emphasizes the persistence of an *accessory* MCA demonstrated by means of cerebral angiography in an elderly lady who suffered an episode of subarachnoid hemorrhage (SAH), due to the rupture of one of two associated inatracranial aneurysms.

CASE REPORT

A 74 year old, right-handed woman, with an eight-year history of untreated arterial hypertension, was hospitalized in our Service on november 19, 2002. Three days earlier, intense suboccipital headache of sudden onset occurred, followed by transient (30 minutes) loss of consciousness and left hemiparesis. The patient was taken initially to another local hospital where a computerized tomography (CT) scan, which turned out to be abnormal, was obtained. Hence, she was transferred to the University Hospital for further evaluation and treatment.

Upon admission, her blood pressure was 160/90 and the rest of the physiological constants, were normal. The patient was disoriented but could attend verbal commands and move freely, if not with her left side. There was moderate neck stiffness, mild left hemiparesis and bilateral extensor plantar response. The pupils were symmetrical and reactive and the fundoscopic examination revealed slight hypertensive arteriolar changes but no subhyloid hemorrhages. A non-contrasted cranial CT scan that relatives carried along (figure 1) showed diffuse SAH, a thin linear clot in the frontal interhemispheric fissure and small amount of intraventricular blood at the bottom of both occipital horns. The ventricular system was slightly enlarged due patient´s age.

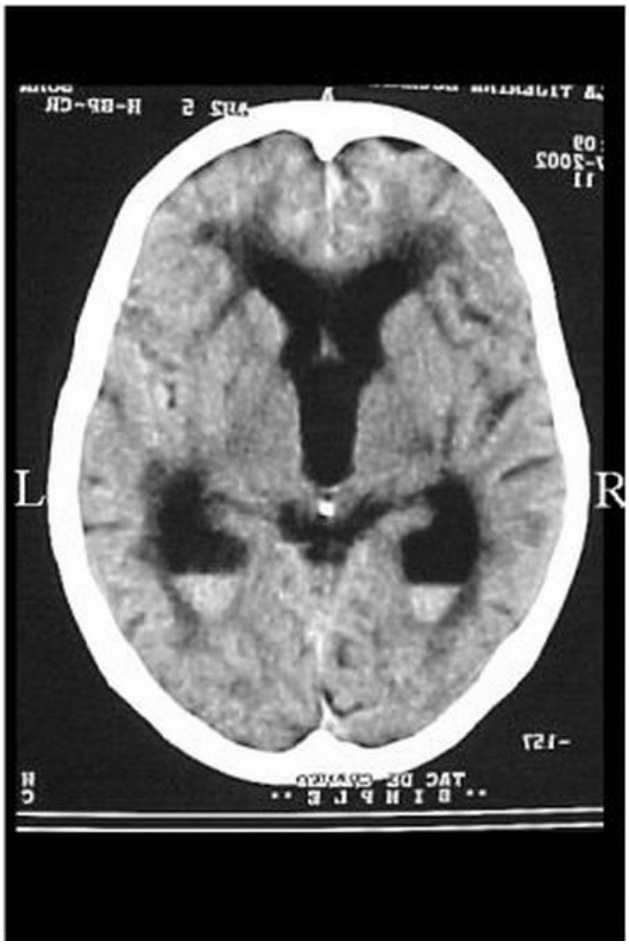

Figure 1. Cranial CT scan shows diffuse SAH with a thin vertical clot in the anterior interhemispheric fissure and small amount of blood in the occipital ventricular horns.

Transfemoral cerebral panangiography was carried out under light sedation the following day. Left ICA injection (figure 2) revealed dominance of ACA-1 with opacification of both pericallosal arteries and a saccular aneurysm arising from the left corner of the anterior communicating artery (ACoA), which was recognized as the SAH´s culprit. In addition, an anomalous arterial channel, roughly paralleling the course of the left MCA, was clearly observed in the anterior-posterior and oblique views. As this second, "twin" like MCA departed from the proximal portion of ACA, extended laterally in the sylvian fissure to arborisation, supplying *pari-passus* thin basal perforating branches, it was readily categorized as an *accessory* MCA. The right ICA

injection (figure 3) demonstrated an hypoplastic A1 segment, 1no pericallosal filling and a *second* aneurysm, emerging at the M1-M2 junction. The proposed microneurosurgical or endovascular treatment was refused.

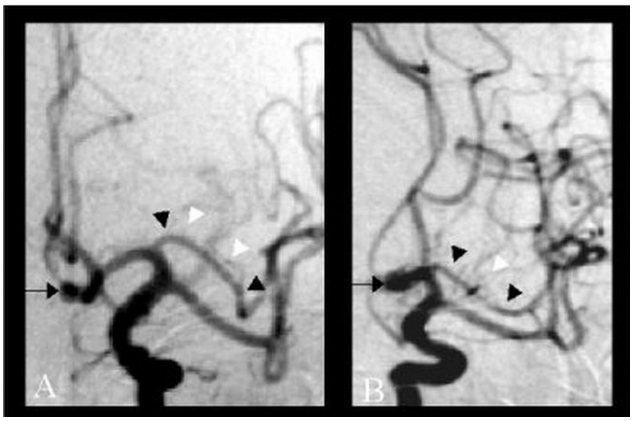

Figure 2. Magnification of left carotid arteriograms, anterior-posterior (A) and oblique (B) views. The accessory MCA (arrowheads) departs from a dominant ACA-1 segment and extends laterally to branching in the sylvian fissure. The abnormal conduit duplicates the course of the MCA and supplies a few medial and lateral basal perforating arteries (white arrowheads). The two pericallosal arteries are opacified. An intracranial aneurysm is shown to depart near the ACA-ACoA function (arrow).

The patient, however, remained hospitalized.

Sudden deterioration occurred two weeks later, the Glasgow coma scale grading declining to 7. After immediate endotracheal intubation and stabilization in the Intensive Care Unit, repeat CT scan of the head disclosed acute hydrocephalus with no trace of recurrent SAH. Actually, the subarachnoid and intraventricular blood observed in the previous CT scan, had vanished. A system for continuous external ventricular drainage was inserted. Regretfully, the patient expired on december 17, 2002, due to nosocomial pneumonia with no neurological improvement. Autopsy request was not granted.

DISCUSSION

An abnormal arterial conduit running back from ACA into the sylvian fissure to replace the orbital branch of the MCA, was observed in a *posmortem* investigation by Blackburn, as early as 1908[2]. The nomination, however, of such irregular recurrent arterial conduits as *accessory* MCA, took place in 1962, by Crompton[3], following the examination of 347 cadaveric brains. In his investigation, 11 examples of double MCA were found, an incidence of 3.2%. The circumstance that 10 of the anomalous arteries departed from ICA and one from ACA did not preclude him from characterizing them all with the same name, attending more to its course than to its different arterial origin. Two years later, Jain[4] published 10 examples of, again, *accessory* MCA, discovered among 300 *postmortem* sylvian arteries dissected, a rate of 3.3%. But at this time, eight of the variants sprang from ACA, and only two from ICA. Finally, in 1973, Teal *et al.* [5], in reporting five additional angiographic examples of double MCA, clarified the confusion by restricting the designation of *accessory* MCA to the anomalous channel departing from ACA, and to term the one deriving from ICA as *duplicate* MCA.

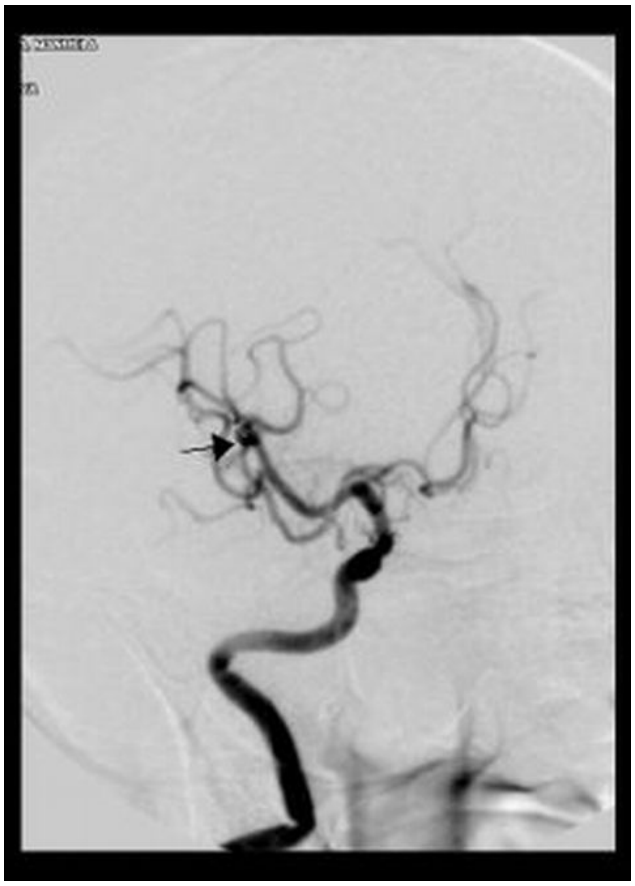

Figure 3. Right carotid arteriogram, oblique view, displays an hypoplastic ACA-1 segment, no pericallocasal arterial filling and a small, second, saccular aneurysm arising at the M1-M2 junction (arrow).

Hence, by universal acceptance, all *accessory* MCAs arise from ACA, most often from ACA-1 [1,4,6-8], but they will at times depart from ACA-2[6,9,10] or ACoA[10,11]. The *duplicate* MCA, in turn, will develop directly from ICA, either at the level of the anterior choroidal artery (AChA)[6], between AChA and ICA end[7,10], or opposite the origin of ACA, as an early ICA bifurcation[1]. As rather unique necropsy findings, an accessory MCA and a duplicate MCA, both, concurred in one occasion in the

same cerebral hemisphere[6]; in one other, the accessory MCA presented bilaterally[4], and in a third instance, an ICA-originated repeat MCA, was associated with reduplication of the circle of Willis[12]. A bilateral accessory MCA has been diagnosed also in a few patients by means of cerebral arteriography[11,13].

More recent *postmortem* microanatomical studies of the sylvian arteries upon unfixed adult brains[1,6] have validated MCA duplication at a rate of 2.9% to 4.0%. In 1968, Handa *et al.* [13] and Krayenbhül and Yasargil[14] first showed such MCA anomalies by *in-vivo* cerebral arteriography. The reported angiographical incidence of accessory and duplicate MCAs combinedly ranges from 0.32%[8] and 1.6% [7], to as high as 4.6% [15] but some of these anomalies will pass clinically unrecognized or unpublished[16]. Proximal *fenestration* (short, partial duplication) is a third MCA anomaly disclosed at autopsy[1,3,6,11] or observed in cerebral angiograms at a rate of 0.26% [1].

MCA abnormalities like these have been described as fortuitous angiographic findings in the investigation of patients suffering from head injury or ischemic stroke[7,8,16], craniopharyngioma or paraganglioma[7], moyamoya disease[10,17] or cerebral arteriovenous malformation[18]. Yet, their most frequent associations are intracranial aneurysms, usually single[1,3,9,14,19,20], at times originating from the abnormal arterial conduit itself [3,15,21]. In a series of 24 patients with SAH and MCA variants drawn from the literature by Umanski *et al.*[1], there were 35 associated intracranial aneurysms diagnosed. In five occasions, an aneurysm arose at the site of the anomaly (20.8%) and in five patients, the aneurysms were multiple (three patients with two, one with four, and one with six aneurysms). Our patient had two aneurysms. Nevertheless, a proclivity for aneurysmal development greater in these patients than in the general population, there seems not to be[1].

The embryological significance of the MCA irregularities is not completely dilucidated as to whether they are the result of early blood flow dynamics or represent a persistent fetal vascular supply. Marubayashi[18] suggests the *accessory* MCA is a residual congenital artery and the *duplicate* MCA represents an early ICA bifurcation. Handa *et al* [19]. See the *accessory* MCA as an hypertrophic recurrent Heubner artery (RHA) for it originates from ACA and supplies basal perforating branches. Yet, the accessory MCA and the RHA may coexist, usually with independent departure. Takahashi *et al.* [22] consider both, the accessory MCA and the RHA, resulting from a primitive anastomotic vascular network supplying the pyriform cortex, with final predominance of one or the other, usually the latter. Furtheremore, Komiyama *et al.* [7] believe the duplicating MCA is a precocious ramification of early MCA branching while Yasargil and Smith[10] prefer not to derive any definitive conclusions in this regard.

In any case, their existence in the brain, in particular the accessory MCA, may serve as significant alternative channels for collateral blood supply reducing a potentially devastating neurological deficit in cases of acute or chronic, embolic or surgical ICA and/or MCA occlusion[16,17,20]. In this eventuality, not rarely, the *accessory* MCA will be demonstrated to fill angiographically from the opposite ICA, across the midline. The preoperatory definition of this type of abnormalities will help the microvascular or endovascular surgeon toward the best treatment of intracranial aneurysms and occlusive cerebral disease.

CONCLUSION

One additional angiographic example of an *accessory* MCA, in this case associated with two supratentorial intracranial aneurysms, is presented. The anomalous MCA in our patient departed from ACA and extended laterally in a recurrent fashion paralleling the course of MCA, providing a few basal perforating arteries before branching.

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