

ARTERIOVENOUS MALFORMATION ASSOCIATED WITH BILATERAL MEDIUM CEREBRAL ARTERY DUPLICATION

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ABSTRACT

Cranial magnetic resonance angiography (MRA) performed in a 23 old patient who presented for a generalized tonico-clonic seizure revealed the association between an AVM and bilateral duplication of MCA. The duplicated MCA did not supply the AVM.

Key words: arteriovenous malformation, duplicated middle cerebral artery, magnetic resonance angiography

INTRODUCTION

Congenital cerebral vascular abnormalities involving the middle cerebral artery (MCA) are unfrequently found. The 3 most common intracranial variations involving MCA are: duplicated MCA, accessory MCA and fenestrated MCA. (1-3) According to the Teal classification (4) when the 2 vessels originate from the distal end of the internal carotid artery (ICA), the condition is called a duplicated MCA. The term accessory MCA is used when the anomalous vessel originates directly from the anterior cerebral artery (ACA). It runs parallel to the course of the MCA and supplies some of the MCA territory: duplicated MCA typically supplies the anterior temporal lobe and accessory MCA typically supplies anterior frontal lobe.

The association between vascular anomalies and an AVM is an unfrequent finding. We present the case of a young man in whom MRA disclosed bilateral duplication of MCA and an AVM supplied by one of the MCA, but not by the duplicated one.

CASE PRESENTATION

A 23 year old patient, with unremarkable past history, was admitted for a generalized tonico-clonic seizure during the night service of the emergency department. The seizure was accompanied by bitten tongue and loss of sphincterian control, and was followed by a confusional state.

The patient was alert and oriented in time and space; he could not recall the events before getting into the ambulance. A right pyramidal syndrome was revealed on physical examination.

An native CT scan disclosed an inhomogeneous area of hypodensity and hyperdensity with calcifications in the left parietal lobe. CT scan with contrast reveals important enhancement (Figura 1, 2)

The CT was suggestive for an AVM that was confirmed by MRI: the inhomogeneous area had a typical aspect of an AVM with small areas of cavernoma and was situated in the left sylvian fissure with extension deep in the white matter of the parietal lobe. (Figura 3, 4)

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MRA in arterial and venous time disclosed a giant AVM, supplied by the left MCA, drained through the superficial venous system consisting in the superior longitudinal sinus and the lateral sinus and through the deep venous system consisting in Galien vein and the straight sinus. (Figura 5, 6, 7, 8). Cavernoma areas were present too, and the AVM

was intricate in the cerebral tissue. The left abnormal vessel was pushed upright forward by the AVM, and branched out vessels for the parietal and frontal area. It did not feed the AVM.

MRA arterial time disclosed bilateral duplication of MCA. (Figura 5 is the better image for the bilateral duplicated MCA)



Figure 1. CT

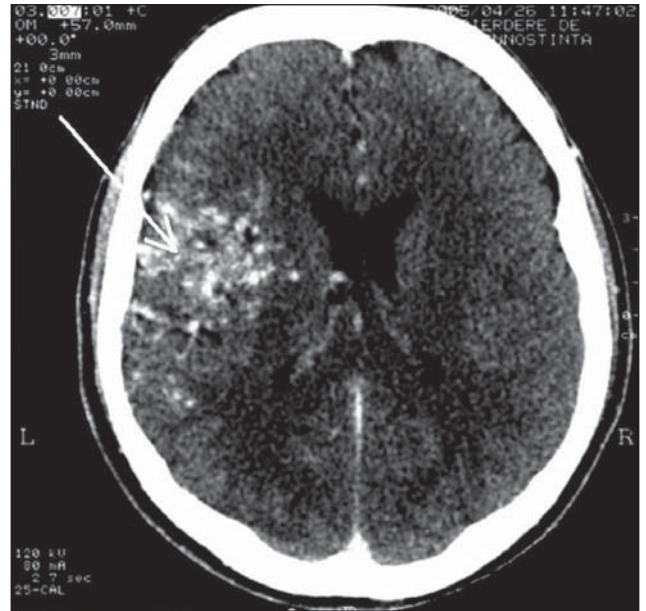


Figure 2. Contrast CT

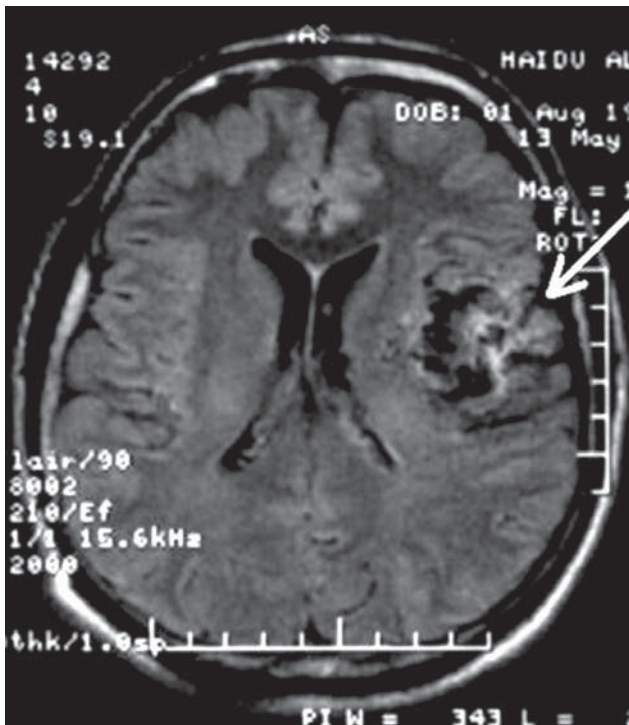


Figure 3. MRI

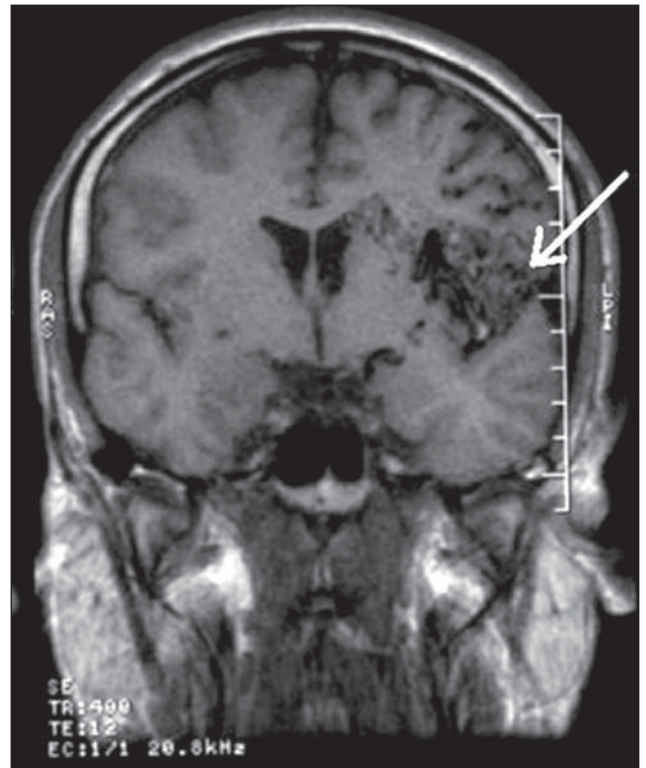


Figure 4. MRI

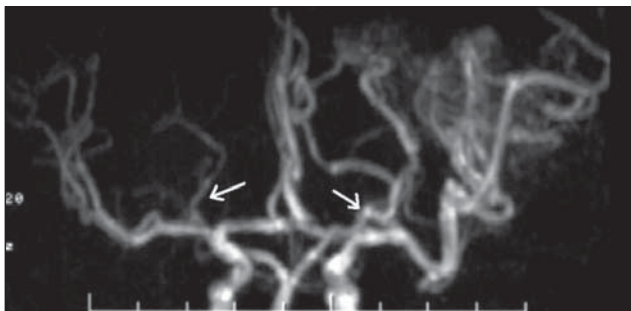


Figure 5. MRA arterial

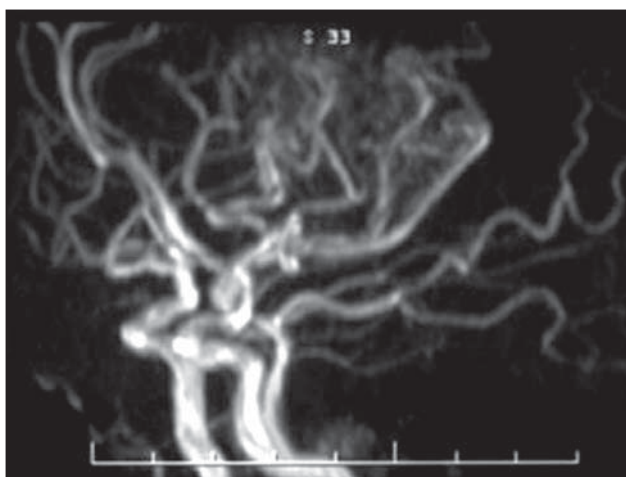


Figure 6. MRA arterial

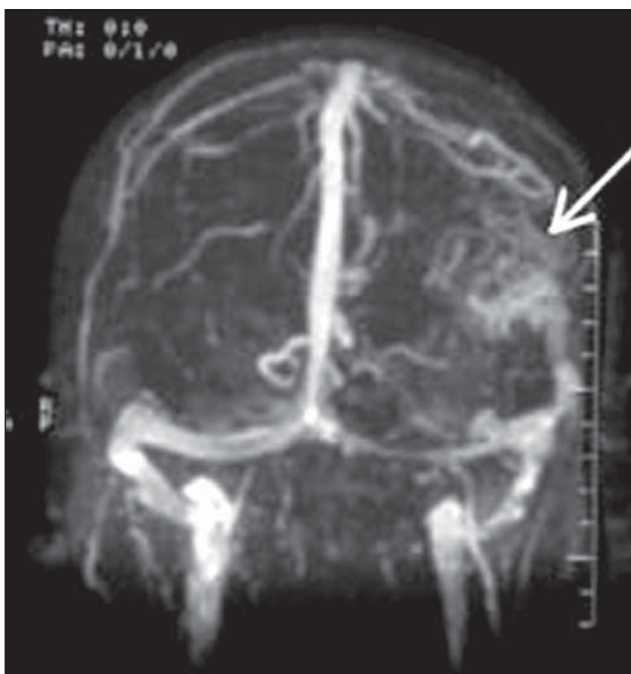


Figure 7. MRA venous

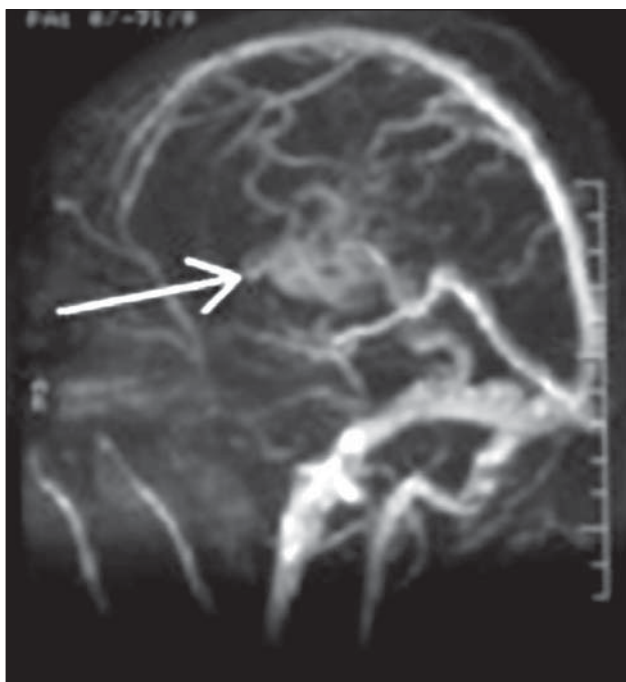


Figure 8. MRA venous

DISCUSSION

Embryological, ACA is considered to be continued by ICA. MCA develops subsequent to ACA, so it can be considered a branch of ACA. MCA is defined in the 7-12 mm embryo as a bud of ICA proximal to ACA. In the 16-18 mm embryo MCA becomes more prominent and its branches irrigate the whole hemisphere. (5,6).

A consensus about the development of MCA anomalies is not established yet. First it was Handa, followed then by other authors (6-10) who said that these anomalous vessels (duplicated or accessory MCA) are hypertrophic and/or modified variants of the recurrent artery of Heubner (RAH).

Teal (4) does not confirm that theory, emphasizing that these arterial variants also have perforating branches, have parallel course with MCA, they co-exist with RAH and they may be multiple.

Manelfe (cited by 3, 11) says that MCA initially is a bud of ICA and ACA which develops lately as a distinct entity. Distinct buds may develop from that primary axis, being situated before or after MCA.

According to that, Manelfe describes 3 types of accessory MCA, type 1 corresponding to the duplicated MCA after Teal, type 2 and type 3 originating proximally or distally in A1 and corresponding to type 1 and 2 after Teal.

The duplicated MCA has a parallel course to MCA and usually supplies the anterior region of the temporal lobe. The accessory MCA distributes to the frontal lobe.

Analysing the vascular abnormality of this patient, respectively the origin of the 2 vessels in the ICA, according to previous reports (1-13), it is duplication that will be defined after the classification of Teal, the one which is accepted now.

The both vessels have initially a parallel course to MCA. The AVM dislocates upwards and forwards the branches of the duplicate MCA, thus they distribute to parietal and posterior frontal areas on the left hemisphere. The duplicated MCA distributes to the temporal lobe on the right hemisphere, just right according to the above mentioned description.

We could not define the deep branches, their sizes being smaller than the sensibility of the method. It was obvious that the duplicated MCA did not supply the AVM.

The presence of these abnormalities of MCA may consist circulatory supply (14), or, on the contrary, lead to lesions with atypical localization, requiring great efforts for diagnosis and treatment. (15)

Numerous cases of association between accessory MCA or duplicated MCA with aneurisms or with moya-moya are reported. (16-21)

The association between these abnormalities and an AVM consists an unfrequent finding as AVM is rarely known to be associated with other types of vascular abnormalities (22,23). We found one similar, but not identical reported case, in which the duplication was unilateral and the duplicate MCA was feeding the AVM (24).

CONCLUSION

The presented case is a rare association: AVM and bilateral duplication of MCA.

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