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Giant pseudoaneurysm originated from distal middle cerebral artery dissection treated by trapping under sensitive evoked potential and motor evoked potential monitoring: Case report and discussion

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Abstract

Background: Dissecting giant pseudoaneurysm of the middle cerebral artery (MCA) is a rare lesion often presenting challenges to neurosurgical teams dealing with this specific pathology. Giant pseudoaneurysm originating from a dissecting distal segment of the MCA treated with aneurysm trapping under motor and sensitive evoked potential monitoring with a successful outcome is presented in the article followed by a brief discussion on the subject.

Case Description: A case of a previously healthy young female patient admitted at the emergency room of Santa Paula Hospital with a history of a sudden headache and syncope, dysphasia, and Grade 4 right hemiparesis due to a large brain hemorrhage secondary to a 25 mm ruptured pseudoaneurysm originated from a distal left MCA dissecting segment is described. Because the patient risked neurological worsening, aneurysm was treated with parent and efferent vessel trapping technique and no changes on the sensitive and motor evoked potential (MEP) from baseline informed on this decision. Hemorrhage was completely drained after aneurysm was secured.

Conclusion: Neurophysiological sensitive and MEP monitoring, on this specific case was a valuable tool and informed on the decision of trapping of this large vascular lesion.

Key Words: Middle cerebral artery, neurophysiological monitoring, pseudoaneurysm, trapping

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INTRODUCTION

Patients with complex middle cerebral artery (MCA) aneurysms – dissecting or giant lesions – may present with intracranial hemorrhage, mass effects, epilepsy or cerebral ischemia; in addition, aneurysm may be incidentally discovered. Often with an unfavorable anatomical morphology of the neck and dome, complex MCA aneurysms present a unique challenge to medical teams.

We describe here a giant pseudoaneurysm case originating from a dissection of the left parietal-occipital branch of the MCA atypically presenting intracranial hemorrhage, surgically treated by trapping of parent and efferent vessels under neurophysiological monitoring. We will present here a brief revision and discussion referencing the literature on dissecting and giant aneurysms and strategies for their treatment.

CASE REPORT

A previously healthy 29-year-old female patient was admitted to the emergency room of Santa Paula Hospital, Sao Paulo, reporting a sudden headache and syncope associated with mild dysphasia and weakness of right arm and leg. Upon admission, she was graded a Glasgow Coma Scale of 13 and a Grade 4 right hemiparesis. According to her husband, she had not suffered any trauma. The patient was hemodynamically stable and reported on regular use of oral contraceptive.

When the patient was submitted to a computed tomography (CT) scan, the following were detected: A nodular hyperdense frontotemporal image anterior to a large left side predominantly temporal hematoma associated to edema and signs of subfalcine and transtentorial herniation, and a midline shift of approximately 6 mm [Figure 1]. An urgent magnetic resonance imaging (MRI) revealed aneurysm of 2, 5 cm in diameter, with partially thrombosed content associated to a large left temporal hematoma [Figure 2]. Digital subtraction angiography revealed dissection of the parietal-occipital branch of the left MCA and large pseudoaneurysm [Figure 3]. Terminal branches of distal MCA were seen emerging from the aneurysm base. In addition, a routine laboratory and cardiologic examination were conducted, yet the results were unrevealing.

Surgery under sensitive evoked potential (SEP) and motor evoked potential (MEP) monitoring was performed on an urgent basis given the risk of further neurological worsening.

A left side large frontotemporal craniotomy was performed along with wide Sylvian fissure opening and complete aneurysm dome and neck dissection. Aneurysm was trapped, and no reduction on neurophysiological parameters from baseline could be noted afterward, thus prompting us not to do a bypass. After the opening of the lesion wall, no bleeding was noted and aneurysmectomy was conducted given the large dome had considerable mass effect on the adjacent brain. Draining of the temporal hemorrhage considerably alleviated regional cerebral edema. Immediate postoperative CT scan showed complete drainage of the temporal bleeding and less midline shift than the previous scan [Figure 4]. Intracranial pressure was monitored and controlled during the first few days, and sedation was lowered on the 3rd day after surgery. A new scan revealed no signs of ischemia. The patient remained in good neurological condition and the previous dysphasia and hemiparesis begun to improve progressively. Control angiotomography showed complete obliteration of aneurysm.

No vasospasm was detected by subsequent transcranial Doppler monitoring done from postoperative day 1. Intense rehabilitation led to complete amelioration of hemiparesis and near total dysphasia improvement, upon discharge from the hospital.

DISCUSSION

Complex aneurysms are defined as having large (10–24 mm in diameter) or giant (diameter ≥25 mm)

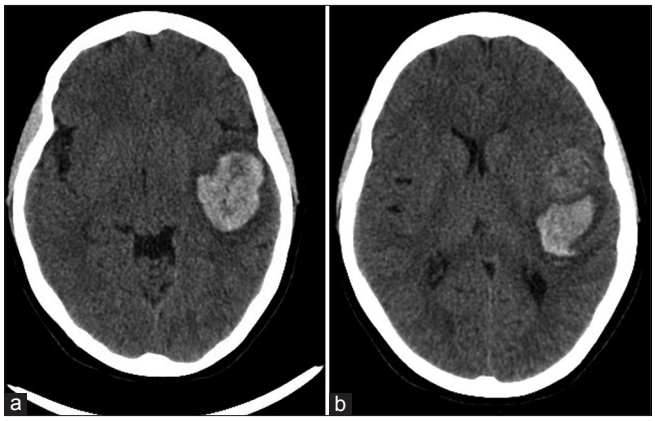

Figure 1: (a) Admission computed tomography scan showing temporal lobe hemorrhage. (b) Admission computed tomography scan: Note nodular hyperdensity in front of hemorrhage

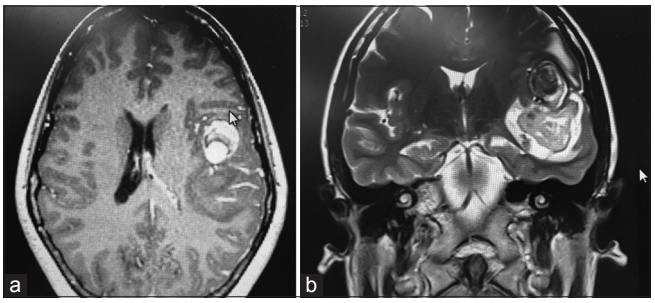

Figure 2: (a) Contrast enhanced axial magnetic resonance imaging: Partially thrombosed pseudoaneurysm. (b) Coronal T2 magnetic resonance imaging of lesion

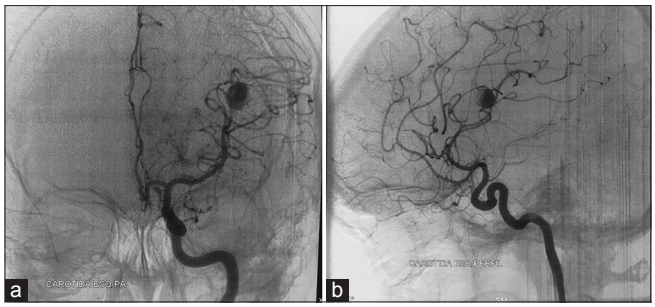

Figure 3: (a) Anteroposterior view of angiography showing distal left middle cerebral artery pseudoaneurysm. (b) Lateral view of angiography

size or nonsaccular morphology (fusiform, dissecting, or serpentine);[23] many of which cannot be treated with direct aneurysm neck clipping or dome coiling.

The literature on dissecting intracranial aneurysms frequently describes them as rare lesions. They are most often related to traumatic events, spontaneous lesions without a clearly identifiable cause are uncommon. They are associated to intracranial vessel dissection, either in the carotid or basilar circulation or on its branches.

When affecting the posterior cerebral circulation, dissecting aneurysms present more often as subarachnoid hemorrhage whereas, in the carotid circulation, the occurrence is more common in the form of cerebral ischemia due to stenosis or vessel occlusion due to thrombus formation. Occurrence in the posterior vessels has been more frequently described and documented. With an increasing number of case reports of cerebral hemorrhage due to these types of lesions in the anterior circulation being more frequently published, we suspect their prevalence may be more significant than we previously thought. A large thrombosed aneurysm associated to MCA distal segment dissection is probably a rare event. In 2002, a Japanese cooperative group reported on a total of 49 dissecting aneurysm in the anterior circulation, 24% of them located on the MCA segment.[12] In 2012, Chuang when reviewing exclusively dissecting MCA aneurysms found 24 case reports.[1] Among these 24 cases, 63% had bleeding presentation and 29% had ischemia presentation whereas two cases were considered incidental finding,[1] which leads us to believe that bleeding might not be this uncommon. In this series, lesions were more often an M1 or M2 pathology type, and distal M3 lesions were unusual.

Considering etiology, dissecting aneurysms may be related to genetic factors, primary and secondary cerebral vasculitis, fibromuscular dysplasia, infection (mycotic aneurysms), traumatic events, atherosclerosis, and hypertension. A report has linked it to migraine syndrome and repeated vascular wall edema with subsequent damage.[18] These lesions probably result from sudden internal elastic lamina disruption and media wall damage,

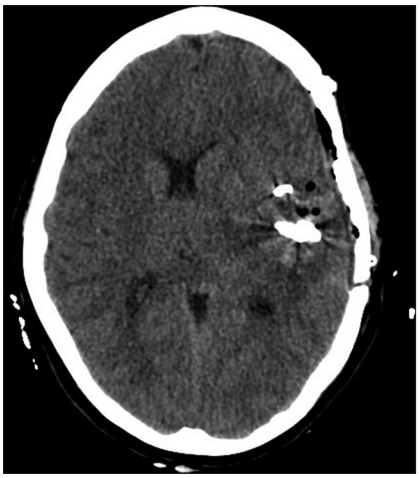

Figure 4: Immediate postoperative scan showing aneurysms clips and complete drainage of hemorrhage and no apparent signs of ischemia

with a plane of dissection either formed between the internal elastic lamina and media or the media and adventitia layers.[10]

Regarding giant aneurysms, occurrence is thought to relate to the same factors that originate smaller saccular lesions. The MCA is the most frequent location for giant aneurysms of the anterior circulation.[9] Giant aneurysms account for as many as 15% of all MCA aneurysms.[8] The largest saccular dilatation ever reported had 12 cm × 8 cm × 6 cm in diameter.[14] In a series of 1344 aneurysms treated, 17.3% were 15 mm or larger and 6.1% were 25 mm or larger.[21] Atherosclerosis possibly play a part on older subjects and enlargement of the wall is thought to occur due to laminar necrosis, thrombus formation within the sac followed by scarring of the wall or repeated hemorrhages, which are subjected to the process of encapsulation and organization.[13] Collagen disease may also be an etiological factor on the development of such large lesions.[13]

Giant aneurysms may produce symptoms and signs that resemble those of a mass lesion, and multiple neurological deficits, seizures, and endocrine disturbances can occur depending on the location.[4,14] Giant MCA aneurysms may also result in chronic, unilateral headaches that might help locate the lesion.[6] A high mortality rate of 65–85% within 2 years (due to rupture or rerupture) has been reported, and aneurysm rupture survivors are often left with severe neurological deficits.[2,7]

Both dissecting and giant aneurysms can be accurately diagnosed using modern neuroimaging techniques. Bleeding and ischemic symptoms can be identified on CT scan and MRI imaging. In the past, when skull radiographs were more frequently used, calcification and bone erosion could be evidenced on patients harboring giant aneurysms.[13] Angiography is of paramount importance for the treatment planning and assessment of

lesions since it may evidence dissection characteristically as the pearl and string sign. Nonthrombosed and thrombosed lesions may appear differently on angiography. Depending on the size of the lesion, neck and parent vessel visualization can be difficult. Angio-MRI usually has a good correspondence with angiographic findings on the posterior circulation but also on carotid segment, as reported on previous studies, and it is a noninvasive technique used for re-examinations when necessary.[12]

Various possibilities exist for the treatment of such lesions, either dissecting aneurysms or giant ones, and none has been well proved by the literature as the gold standard. de Divitiis *et al.* mentions that if there is a high risk of rebleeding (growing dissecting aneurysm, giant dissecting aneurysm or dissection associated with uncontrolled hypertension), direct treatment should be indicated.[3]

In general, lesions presenting with bleeding should be treated early to prevent rebleeding on patients on a favorable neurological condition. An estimated 30% chance of rebleeding of posterior circulation dissecting aneurysms has led some authors to consider a more urgent intervention when considering lesions on anterior circulation.[1] There is a paucity of information regarding the timing of intervention on giant partially thrombosed dissecting aneurysms of distal MCA segment. We opted for an early intervention based on the risk of further neurological worsening since the patient carried a large temporal hematoma risking internal uncal herniation.

Whenever possible, treatment strategies should be decided by a cerebrovascular team comprised both by neurovascular surgeons and endovascular specialists. Definite aneurysm treatment generally consists of one of the following techniques depending on each case: Endovascular therapy, parent vessel occlusion, neck reconstruction and clipping, wrapping with wall reinforcement, and trapping with or without bypass procedures. We believe direct aneurysm neck clipping and exclusion with aneurysmectomy in cases of giant lesion with mass effect to be desirable, although not always possible, especially in the case of fusiform dilatation and branches originating directly from the aneurysm wall. During surgery, superficial temporal artery branches should be preserved if a bypass is anticipated and needed due to parent and efferent vessel occlusion or ischemic symptoms.

For saccular aneurysms with a broad neck or giant size, we often use low-flow electrocoagulation to shrink the aneurysm wall or to mold the neck after temporary parent artery clipping. This is a minor procedure, but it may transform an unclippable aneurysm into a clippable one. For giant aneurysms with severe intraluminal thrombus, thrombectomy is usually required to facilitate direct clipping.[15,19] For previously coiled aneurysms, an evacuation of the coils under temporary parent artery occlusion is usually performed before we can apply a clip on the neck of aneurysm.[16,22]

Brain relaxation can be achieved by administration of mannitol (1 g/kg) and dexamethasone (5 mg) or cerebrospinal fluid drainage through dissection into a subarachnoid cistern. When necessary, parent artery temporary occlusion, mild hypothermia, barbiturate, or propofol have been used to increase tolerance to ischemia.

During our surgery, aneurysm was found to be an unclippable one because of its dissecting morphology. The proximal and distal M3 segments were temporary occluded. Intraoperative MEP and SEP showed no change after 30 min of occlusion during the aneurysm dissection. Therefore, the aneurysm was trapped, and an aneurysmectomy was performed. The patient recovered well without additional neurological deficits despite any revascularization procedure. Data on the use of MEP during aneurysm clipping indicate that preserved MEPs at the end of the procedure usually correspond to normal neurological postoperative examination. Mild motor deficit, in the case of preserved MEP, is usually transient and improves within a week postsurgery. Transient MEP loss is followed by a mild to transient weakness, and permanent loss of MEP is always followed by severe motor impairment, usually with early ischemic signs on immediate postoperative scans.[20] Neither MEPs and SEPs changes from baseline nor early signs of ischemia on immediate post surgery scan were noted on our patient. Sciubba *et al.*, however, have reported on a patient operated with preserved intraoperative MEPs followed by postoperative ischemic damage[17] although without additional neurological impairment and there are also cases clearly related to postsurgical adverse events (hypotension, cerebral edema) leading to ischemia.[20] Combined use of intraoperative neurophysiologic monitoring and preoperative parent vessel occlusion test, when feasible, may also be the option in cases where vessel occlusion is anticipated and might point to a bypass. Nussbaum *et al.* report on a large series of 60 peripheral complex aneurysms, and revascularization was carried out on cases where sacrifice of the parent vessel was necessary. The article does not mention the use of neurophysiologic monitoring during surgical procedures.[11]

The adequacy of the treatment and patency of the parent vessels after clipping may be analyzed intraoperatively using indocyanine green fluorescence angiography and/or Doppler flow measurements. Intraoperative SEP monitoring and MEP monitoring is routinely performed to detect potential injury to neurological functions. Changes in SEP or MEP have been managed by increasing the blood pressure with pressor agents or even clip repositioning according to the surgeon judgment.

Wrapping procedures consist of aneurysm wall reinforcement using muscle, cotton, Silastic sheet, Teflon, and adhesives. Figueiredo *et al.* have reported nine cases of clip wrapping technique on complex aneurysms with no rebleeding events on an average of 2 years follow-up,[5] although other authors have not obtained same successful results experiencing considerable rebleeding rates.

It is beyond our present objective to discuss endovascular therapy (coiling, stenting, diversion flow devices) of dissecting and giant aneurysms. Although there are still major limitations associated with endovascular therapy of MCA aneurysms and of giant aneurysms, in particular, a multidisciplinary discussion should always be encouraged when assessing such complex lesions.

CONCLUSION

A case of an atypical giant aneurismal lesion originating from a dissection of a distal branch (M3) of MCA was reported. Because presentation was associated with a large temporal lobe hematoma and the patient risked neurological deterioration, early surgical drainage, and trapping of aneurism was successfully conducted, and progressive improvement could be verified. Preoperative analysis of the morphology of aneurysm, vascular anatomy, hemodynamic characteristics, and intraoperative MEP and SEP monitoring was a decisive factor in choosing appropriate surgical strategies. No intraoperative MEP and SEP changes informed the decision to occlude parent and efferent vessels. The patient experienced a favorable outcome (Glasgow Outcome Scale 5), and no ischemic lesions were evident on imaging controls.

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Conflicts of interest
There are no conflicts of interest.

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