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Endovascular treatment of fusiform A2 aneurysm with parent artery occlusion

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Abstract

Background: A2 aneurysms are rare with a reported incidence of <1% of the intracranial aneurysms. These aneurysms are located between the anterior communicating artery and genu of the corpus callosum. Fusiform aneurysms in this location are even rarer and we present one such case of fusiform A2 aneurysm treated with endovascular technique.

Case Description: In this report, we present a case of ruptured fusiform A2 or proximal pericallosal artery aneurysm in a middle-aged female who presented with subarachnoid hemorrhage. She subsequently underwent endovascular parent artery occlusion, and post-procedure angiogram showed good pial collaterals filling the distal territory. She developed transient lower limb weakness which improved over the next 24 h with supportive inotrope management to maintain adequate cerebral flow.

Conclusion: We report a rare unique case of ruptured fusiform proximal pericallosal artery aneurysm. Endovascular treatment of this type of aneurysm is a feasible method and can be considered as an effective alternative to surgical technique.

Key Words: A2 aneurysm, coiling, fusiform aneurysm



INTRODUCTION

Anterior cerebral artery (ACA) aneurysms are divided into five groups which include proximal ACA or A1 aneurysms, anterior communicating artery aneurysms, proximal pericallosal artery or A2 aneurysms, classical pericallosal or A3 aneurysms curving around the genu, and A4, A5 aneurysms which include distal branch aneurysms.

A2 aneurysms, otherwise known as proximal pericallosal artery aneurysms, are very rare with an incidence of 0.2-1% of all intracranial aneurysms.^[5,6,13-15]

The A2 aneurysms are located between the anterior communicating artery complex and the genu of the corpus callosum on the frontobasal branches.

Fusiform A2 aneurysms are extremely rare with only few reports described till date.^[8,12]

Most of these aneurysms arise at the origin of the frontopolar artery and are usually saccular. We present a rare case of a fusiform proximal pericallosal artery aneurysm arising from the distal part of A2 prior to the origin of callosomarginal artery and treated with endovascular coil occlusion.

CASE REPORT

A 32-year-old female presented with sudden onset of severe headache and neck pain and altered sensorium. Magnetic resonance imaging (MRI) showed hyperintensities in the subarachnoid space predominantly in the anterior interhemispheric fissure on fluid attenuation inversion recovery T1 images [Figure 1]. Digital subtraction angiogram (DSA) showed fusiform elongated aneurysm with a small bleb in the left distal A2 segment and severe vasospasm in bilateral A1 segments of the ACA [Figure 2]. Based on the decision of our multidisciplinary team, endovascular coil occlusion along with the parent artery was planned.

Procedure

Under general anesthesia, via tansfemoral approach, a 6 F guiding catheter was placed in the left petrous internal carotid artery. Thereafter, echelon 10 microcatheter (eV3; Neurovascular, Irvine, CA, USA) over transcend micro wire (Boston Scientific, Freemont, CA, USA) was advanced gently into the distal A2 aneurysm. A total of five Axium coils (eV3; Neurovascular) were placed and the aneurysm occluded along with the distal A2 segment [Figure 3].

Post-procedure angiogram [Figure 4] showed complete exclusion of the aneurysm from the circulation with



Figure I: Axial TI weighted image showing the bleed in the interhemispheric fissure (a) and corpus callosum(b)



Figure 3: Left oblique view of left ICA angiogram showing the fusiform aneurysm in A2 (arrow in a). Microcatheter advanced into aneurysm under roadmap guidance and aneurysm packed with coils (arrow in b)

parent artery sacrifice and the distal ACA territory was seen filling via the pial collaterals from the middle cerebral artery.

On extubation, patient developed weakness of right lower limb (power = 2/5). She was then kept on dopamine infusion to achieve mean arterial pressure (MAP) of >90 mm Hg in order to maintain adequate cerebral blood perfusion, thus facilitating good revascularization via good pial collaterals to retain distal ACA supply. Her weakness improved completely over the next 24 h and she was discharged on the 9th postoperative day. On 1 month clinical follow-up, she did not have any complaints and her neurological examination was normal.

DISCUSSION

A2 aneurysms (proximal pericallosal artery aneurysms) are rare with a reported incidence of 0.2-1%. These aneurysms represent 5-22% of all Distal anterior cerebral artery (DACA) aneurysms.^[5,6,13-15]

These aneurysms are usually saccular and have high tendency to rebleed. Hence, early treatment and exclusion of the aneurysm by surgery or endovascular method is required.^[1,4,8]

Fusiform aneurysms in this location are very rare and are characterized by pathological tortuosity and elongation. Fusiform aneurysms are more commonly seen in the posterior circulation than in the anterior circulation.^[17]



Figure 2: Digital subtraction angiogram of the left internal carotid artery showing the fusiform aneurysm in the proximal pericallosal artery (white arrow in a) and vasospasm in the proximal AI (black arrows in a). Right ICA angiogram showing severe vasospasm in the left anterior cerebral artery (arrows in b)



Figure 4: Post coiling angiogram showing the coil mass (black arrow in a) and proximal stump of A2 (white arrow in a). Capillary phase showing good pial collaterals filling the ACA territory after the coil mass (arrow in b)

Only a few case reports are available in the literature describing fusiform A2 aneurysm.^[8,12]

Lehecka *et al.*, in their review on microsurgical treatment of A2 aneurysms, found 35 A2 aneurysms out of a total of 4253 intracranial aneurysms and 21 were ruptured. Only one case of fusiform A2 aneurysm was described in their entire series. Animal study was done on ACA reconstruction by various authors. Yokoh *et al.*^[16] described ACA reconstruction in their study of three cases, where they used end-to-side anastomoses between both A2s and side-to-side anastomosis between A1 and A2. The site should be carefully selected due to the presence of perforating vessels in this location. These complex procedures are only possible in experienced hands.

Multiple aneurysms are usually seen in association with A2 aneurysms of DACA.^[5,7]

Lehecka *et al.* in their series found 46% of the patients had at least one additional aneurysm.^[8] But in our patient, we did not find any other additional aneurysms in the intracranial cerebral vasculature.

Intracerebral hemorrhage (ICH) is another common presentation seen in these patients, since these aneurysms bleed frequently into the adjacent brain parenchyma and the presence of bleed is often associated with intraventricular extension.^[9]

ICH has a negative implication on prognosis because of the limited subarachnoid space adjacent to the aneurysms.^[9]

The aneurysms in this location pose a difficult challenge to the surgeon because of the narrow space of interhemispheric fissure, dense adhesions between gyri, and difficulty in identifying the neck in such a narrow space.

Involvement of the perforating arteries from anterior communicating artery to A2, as well as frequent arterial anomalies makes even the microsurgical treatment a difficult option in these cases. Surgical techniques of wrapping, proximal occlusion, excision, trapping, and parent artery occlusion with proximal bypass and reconstruction have been described in literature for these aneurysms.^[7,12] Several authors reported anterior interhemispheric or bifrontal basal anterior interhemispheric approach to achieve proximal occlusion.^[2,10]

Endovascular technique by coiling of these aneurysms has been described by some authors.^[3,11] Technical difficulties in these cases include reaching the distal vessels with microcatheter and achieving stable position for coiling. Presence of severe vasospasm is another factor that hinders the advancement of microcatheter to the aneurysm as seen in our case. With the recent and latest advancements in the hardware, small diameter and flow-guided catheters are available, which are more favorable in reaching such distal locations, thus making the endovascular method still a feasible option. Because of the associated severe vasospasm of the proximal arteries and fusiform nature of the aneurysm in the present case, we chose endovascular method over surgery.

Distal infarction is usually prevented by good and adequate leptomeningeal and pial collaterals in these cases. In our case, in spite of good pial collaterals on the post coiling angiogram, patient developed weakness of the right lower limb after the procedure. We kept her on dopamine infusion (0.5 mcg/h) in order to maintain adequate cerebral perfusion and facilitate revascularization of the distal ACA territory. Because of the induced hypertension, there is increased collateral flow as in our case. The development of weakness in spite of good collaterals can be partially attributed to the severe vasospasm. Her deficit gradually improved over the next 24 h with full recovery of the power in the right lower limb. Despite the risk of distal infarction, endovascular parent artery occlusion is the preferred treatment because of high mortality in these aneurysms if left untreated.

CONCLUSION

In the present paper, we describe a rare case of ruptured fusiform A2 aneurysm treated successfully by endovascular parent vessel occlusion. To our knowledge, this is the first reported case of this entity treated by endovascular technique.

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