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Abstract

Background. Aneurysms of the distal anterior cerebral artery (DACA) are uncommon; they often form near the pericallosal-callosomarginal junction and are typically small. To our knowledge, giant DACA aneurysms developing from the more distant parts of the anterior cerebral artery (ACA), A4-5, have been described only once in the literature.

Case description. A 66-year-old gentleman reported with a brief loss of consciousness followed by weakness in his right lower leg. The patient was admitted with a Glasgow Coma Score (GCS) of 15. A computed tomography (CT) scan of the head revealed a left hyperdense mass in the frontal parasagittal supracallosal region. Contrast MRI revealed a heterogeneously enhancing mass measuring 35x30x25 mm. CT angiography (CTA) revealed a small saccular aneurysm on the posteromedial aspect of the mass, perpendicular to the vertical plane of the coronal suture, corresponding to the A4-A5 junction of the left ACA. Through a left paramedian craniotomy, a modified anterior interhemispheric approach that was more posterior than the conventional projection was performed. A giant partially thrombosed was found. The aneurysm was resected, and the neck was reconstructed using four clips placed on top of them to enhance the clipping force over any remaining thrombus. The patient recovered as expected and was neurologically intact three months later. Conclusion. Giant distal anterior cerebral artery (DACA) aneurysms found in the A4-A5 segment represent a pathologically uncommon phenomenon. Due to the rarity of giant aneurysms at this location, their reporting is important to inform meticulous pre-operative planning.



INTRODUCTION

The distal anterior cerebral artery (DACA) represents all segments of the anterior cerebral artery (ACA) distal to the anterior communicating artery. DACA aneurysms constitute up to 6% of all intracranial aneurysms (7, 10, 12, 13, 16, 17, 19, 21, 23, 29, 30, 31). Most of these aneurysms arise from relatively "proximal" segments of the ACA; mainly the pericallosal-callosomarginal junction (69-82%). DACA aneurysms arising from the more distal A4 and A5 segments of ACA are very rare; representing less than 0.6% of all intracranial aneurysms (7, 13).



Figure 1. Pre-operative imaging. Axial sections of non-contrast head CT scan (**A**) as well as T1 (**B**), T2 (**C**), FLAIR (**D**), and gadoliniumenhanced T1 (**E**) MRI show a left frontal parasagittal (supracallosal) mass that appears hyperdense on CT and heterogenous on MRI, with contrast enhancement of the posteromedial part of the lesion.



Figure 2. Pre-operative CT angiography with **(A)** Axial section, **(B)** sagittal section, and **(C)** 3D reconstruction. A superiorly-directing aneurysm (8x4 mm) can be seen at the A4-A5 junction, with 2 distal A5 branches arising from the neck of the aneurysm. The location of this aneurysm corresponds to the posteromedial enhancement of the mass on MRI.



Figure 2. Intraoperative images for a pericoronal anterior interhemispheric approach. A: Initial exposure showing the large dome of the aneurysm (D), which is opened for internal decompression and thrombectomy. B: The borders of the giant dome of the

aneurysm are outlined in yellow with an incision in the center for internal decompression and thrombectomy. C: Following internal decompression of the thrombosed aneurysmal dome, the pericallosal artery (A4) and the distal branches (A5) can be seen, as well as the neck (N) and dome (D) of the intraluminal portion of the aneurysm, prior to the application of clips. Hemosiderin staining can be seen deep within the interhemispheric fissure, denoting old hemorrhage from the aneurysm.

FC; frontal cortex, D; aneurysmal dome, F; falx cerebri, N; aneurysmal neck, A4, A5; segments of pericallosal artery.

Giant DACA aneurysms (those measuring more than 25 mm in their greatest diameter) are extremely rare and mostly fusiform in nature. To the best of our knowledge, saccular aneurysms of the A4-A5 segments have only been reported once in the literature (4). Giant partially thrombosed aneurysms usually have been proposed to result from accumulative intramural bleeding, enlargement, and thrombosis (11). Therefore, endovascular treatment can often be ineffective, as it only eliminates the intraluminal portion of the aneurysm, leaving the mural portion liable to more bleeding, enlargement, and even rupture (2). This makes microsurgical management of giant partially thrombosed A4-A5 aneurysms more appealing, and raises several surgical challenges owing to their location posteriorly within the anterior interhemispheric fissure, the configuration of their domes, involvement of proximal and distal vessels within their base, and the mass effect which can significantly alter the anatomy of this region (11-13).

All of the aforementioned highlights the importance of this paper, in which we report the second case of an idiopathic, giant, saccular, partially thrombosed A4-A5 DACA aneurysm, discussing the treatment nuances of such extremely rare lesions.

CASE DESCRIPTION

A 66-year-old male with a history of a prior ischemic stroke and infrequent seizures on treatment presented with sudden temporary loss of consciousness, followed by right lower limb weakness. He had complained of a poorly localized headache for the past few months with no other symptoms. On admission, the patient had a Glasgow Coma Score (GCS) of 15 and his neurological examination was normal apart from weakness of the right lower limb, medical research council (MRC) grade 4.

Head computed tomography (CT) scan revealed a left frontal parasagittal supracallosal hyperdense mass with relative ventriculomegaly (figure 1A). Magnetic resonance imaging (MRI) of the brain showed a left extra-axial heterogenous lesion measuring 35x30x25 mm, with contrast enhancement of its postero-medial part (figure 1B-1E). CT angiography (CTA) showed a small (8x4 mm) saccular aneurysm perpendicular to the vertical plane of the coronal suture, corresponding to the A4-A5 junction of the left ACA (figure 2). Digital subtraction angiography was not performed as modalities were not available. This aneurysm corresponds to the contrast-enhancing part of the lesion. A pre-operative list of differentials for the mass was made to help with planning for the procedure, and included cavernous malformation, hematoma, neoplasm, and lastly, or a thrombosed part of an aneurysm.

The patient was operated via a left paramedian craniotomy bisected by the coronal suture, and a modified anterior interhemispheric approach that was more posterior than the classic projection, to target the more posterior A4-A5 junction. Splitting of the fissure revealed a yellowish, solid wall of a thrombosed part of the aneurysm, corresponding to the giant mass on preoperative imaging (figure 3A). A rupture site on the lateral surface of the dome (the part of the dome facing the cerebral hemisphere). The fissure also showed thick adhesions and hemosiderin staining, indicating previous rupture of the aneurysm. The thrombosed part was opened, the thrombus evacuated and the mass internally decompressed to allow easier exposure of the active part of the aneurysm and identification of proximal and distal vessels (figure 3B). Proximal control of the ipsilateral A4 was gained, and the aneurysm was resected, followed by reconstruction of the neck with 2 opposing tandem clips and 2 additional clips stacked over them to reinforce the clipping force over any residual thrombus. Patency of proximal and distal vessels was visually confirmed by microdoppler. No intraoperative angiography was available in the facility.

Postoperatively, the patient was fully conscious with no new neurological deficits. A post-operative CTA showed complete occlusion of the aneurysm, with preservation of the distal branches of the ACA. The patient was put on antiepileptic medications and discharged 7 days post-operatively, One- and 3month follow-up appointments revealed a neurologically intact patient, with complete resolution of the headache and no seizure activity.

DISCUSSION

The ACA is anatomically divided into 5 segments; A1 through A5. The first "pre-communicating" segment arises at the bifurcation of the internal carotid artery, and ends at the anterior communicating artery. The second segment extends to the region between the rostrum and genu of corpus callosum, and its continuation curving anterior to the genu is the A3 segment, which ends at the rostral part of the body of corpus callosum. A4 and A5 segments comprise the horizontal portion of the ACA, and are demarcated by the virtual plane of the coronal suture. A2 to A5 segments are collectively referred to as DACA (12, 13).

The majority of DACA aneurysms (69-82%) are located within the A3 segment, usually around the callosomarginal-pericallosal junction. DACA aneurysms of the A4-A5 segments are rare, representing 5-20% of DACA aneurysms, and less than 0.6% of all intracranial aneurysms (7, 10, 13, 16, 17, 19, 21, 23, 29, 30, 31). A4-A5 aneurysms arise anywhere along the horizontal portion of the ACA back towards the spleneum of the corpus callosum, and the distal cortical branches that arise from the termination of that segment (12).

On the other hand, giant cerebral aneurysms are in themselves a rare occurrence, representing about 5-8% percent of all intracranial aneurysms (13). With a predilection for the posterior circulation, they tend to arise in large vessels, such as the basilar tip, and are usually partially or completely thrombosed (15, 27). A possible explanation for this predilection is the increased flow rate at these locations, which increases the shear forces acting on the vessel wall, increasing the risk of damage and microdissection (32). Such vascular injuries accumulate damage on the vasa vasorum of the vessel, causing repeated intramural bleeding and progressive swelling of the aneurysm into giant sizes (2). Additional factors such as trauma or infection also play a role in the pathogenesis of these giant aneurysms (13). There are less than 40 cases of giant DACA aneurysms reported in the literature. The majority of those are fusiform in nature, and none are located at the A4-A5 segments (5, 12, 14, 15, 20, 25). A possible explanation of this scarcity would be the decreased flow rate at the distal cerebral circulation, reducing

the wall shear stress on the arterial walls and making damage, dissection, and formation of giant aneurysms less likely (8, 18, 32). As such, the finding of an idiopathic, giant saccular aneurysm at this location is surprising, counter-intuitive, and raises questions regarding the possible underlying pathomechanisms of these lesions.

The natural history of giant thrombosed aneurysms tends to differ from that of intracranial aneurysms in general. In many cases, they present due to their size as mass lesions compressing adjacent structures and causing subsequent neurological deficits, as was the case in our patient (11, 13). However, according to the International Study of Unruptured Intracranial Aneurysms (ISUIA) trial, 50% of giant intracranial aneurysms tend to rupture within 5 years of presentation (28). It is worthwhile to mention that the ISUIA does not report on the presence of intramural thrombosis within these giant aneurysms, which might be an important determinant of the risk of rupture (11).

The pathomechanism responsible for the formation of giant partially thrombosed aneurysms implies that bleeding from those lesions is often extraluminal (2). This entails that endovascular management of giant partially thrombosed aneurysms can be ineffective (14, 27). Endovascular coiling can obliterate the lumen of the aneurysm, but the aneurysm continues to grow despite a patent lumen as their growth is related to intramural hemorrhage from the vasa vasorum (2, 14). Moreover, coiling DACA aneurysms in general poses a challenge compared to other aneurysms, given the distal location of these lesions (12). As such, resection of these lesions with neck reconstruction or trapping with bypass may provide the best outcomes (15, 22, 31).

The scarcity of giant saccular DACA aneurysms in general, all the more so of giant A4-A5 aneurysms, makes for little experience in the surgical management of such complex lesions. DACA aneurysms are treated via an anterior interhemispheric approach (12). The interhemispheric fissure presents a very narrow and challenging surgical corridor, leaving little room for instrument manipulation and limiting the possible angles for application of clips (12, 13). To complicate things, A4-A5 aneurysms, laying way posterior to the genu of corpus, are difficult to identify in the fissure due to lack of any reliable landmarks at this location (12). This posterior location also means that the classic anterior interhemispheric approach needs to be modified, to project more posteriorly and reach the lesion.

We recommend creating a paramedian craniotomy centered around the coronal suture, to ensure direct access to the A4-A5 junction. Additional factors also affect the anatomy of the fissure, such as the presence of a large intracerebral hematoma displacing the structures, subarachnoid hemorrhage causing thick adhesions within the fissure, and the variation in the anatomy of A4-A5 segments and their branches (12, 13, 26). Fortunately, internal thrombectomy and decompression of the giant thrombosed part in our case was used reliably to reach the active part of the aneurysm and identify proximal and distal vessels. An alternative would be the use of neuronavigation to help guide the surgeon into the lesion (15).

Resection of the thrombosed part of the aneurysm ensures the arterial dissection and potential intramural space is treated (2, 11). Simple clipping is sometimes difficult in these cases, due to their wide-base and the presence of atherosclerotic plaques at the neck, preventing full closure of the clip (9). This can be overcome with multiclipping techniques like booster clips and tandem clipping, as well as techniques like clip compression and microendarterectomy (3, 9, 24). In our case, clip reconstruction of the neck was possible, due to thorough evacuation of the thrombosis, absence of calcification at the base of the aneurysm, and favorable configuration of the pericallosal artery, which allowed for the application of the clips without impinging proximal and distal vessels. When neck reconstruction is difficult, microsurgical trapping of the parent vessel with intracranial-intracranial bypass can be attempted. This technique ensures cessation of blood flow both into the wall and the lumen of the aneurysm while maintaining blood flow to distal vessels.

Many bypass techniques for the reconstruction of the ACA and its branches have been studied (30). However, these intracranial-intracranial bypass techniques require a high level of surgical dexterity, practice, and experience, and carry a possibility of failure and complications, particularly when dealing with small-caliber vessels such as those of the distal ACA (1, 6, 12, 13).

Giant partially thrombosed aneurysms of the A4-

A5 segments of the ACA are extremely rare lesions. We report the second such case in the literature. The intramural -rather than intraluminal- nature of these lesions makes them poor candidates for endovascular therapy. The anatomical disruption they cause within the anterior interhemispheric fissure makes the already difficult task of locating them more daunting. Excluding these lesions from the circulation without compromising proximal and distal branches of the pericallosal artery is challenging, and requires a comprehensive knowledge of the pertinent anatomy, as well as experience in microvascular procedures like neck reconstruction and intracranial-intracranial bypass techniques.

CONCLUSIONS

Giant partially thrombosed A4-A5 aneurysms are an extremely rare disease entity. Their pathological mechanisms are likely to be different from small saccular aneurysms, and their location poses unique surgical challenges. Surgical management must be adjusted to deal with these challenges and ensure the best outcome.

ABBREVIATIONS

DACA: Distal anterior cerebral artery. CT: Computed tomography. MRI: Magnetic resonance imaging. CTA: CT angiography. GCS: Glasgow Coma score. MRC: Medical research council.

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