Dissecting Aneurysm of the Anterior Cerebral Artery Presenting with Thrombo-Embolic Complications

A Case Report

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Summary

A dissecting aneurysm of the anterior cerebral artery is a relatively rare disorder. A patient is presented with mild symptoms due to thrombo-embolic complications from a dissecting aneurysm of the pericallosal artery. The patient had a good outcome after conservative treatment. A review of the literature is presented.

Introduction

Intracranial dissecting aneurysm is a rare clinical entity, although the number of recognized cases has increased. Most cervicocerebral dissecting aneurysms are located in the vertebrobasilar system, and those found in the anterior cerebral circulation are mainly located in the middle cerebral artery. Dissecting cerebral aneurysms strictly confined to the anterior cerebral artery are rare. This lesion presents with two different clinical presentations; subarachnoid haemorrhage or ischemia. A case of a young female with a dissecting aneurysm of the pericallosal artery, presenting with mild symptoms is described.

Case Description

A 22-years-old female patient presented with a slight paresis of the right leg, and a mild expressive aphasia without other neurological

symptoms. In the months before presentation she had experienced a few short periods with a "heavy" feeling in her right leg. She was not known with medical problems and there was no preceding trauma. Neurological examination revealed no other abnormality. Because multiple sclerosis was suspected an MRI was performed.

The T2-weighted images showed a mixed lesion in the left anterior frontal lobe with hypodense rim, surrounded by cerebral edema (figure 1). The T1-weighted images showed a high signal in the periphery of the lesion and after gadolinium injection the lesion showed enhancement (figure 2, 3). A sagittal reconstruction with a thick slice showed the relation between the lesion and the pericallosal artery (figure 4). The diffusion weighted images showed areas with increased signal intensity in left anterior cerebral artery territory (figure 5). Because at this point a vascular lesion was suspected an MRA and DSA were performed.

The MRA and DSA showed a normal anatomy of both A1 and A2 segments as well as a normal anterior communicating artery (figure 6, 7). Both right and left site have a prominence of the orbital frontal and frontal polar distribution, the left frontal polar artery having almost the same diameter as the pericallosal artery. The callosomarginal artery is not developed on either site.

Figure 1 T2-weighted axial image shows a heterogeneous lesion in the anterior left frontal lobe, with a hemosiderin rim and a central thrombus. The lesion is surrounded by cerebral edema.

Figure 2 T1-weighted axial image shows the high signal due to the methemoglobin in the thrombus.

The left pericallosal artery has an elongated aspect with two loops and an irregular contour. On the level of the genu of the corpus callosum the vessel widens and there is a slow filling of an aneurysm with an irregular contour, suggestive of intraluminal thrombosis. The distal pericallosal artery was dilated. The flow in the pericallosal artery is very slow, in the venous phase the distal pericallosal artery is still enhanced. No crossover of the right pericallosal branches was seen.

The initial symptoms subsided and only an attention deficit remained, which gradually diminished. The diagnosis of fusiform dissecting aneurysm of the anterior cerebral artery, with a focal partial thrombosed aneurysm, and thrombo-embolic complications was made. Treatment options were discussed and it was proposed to test for collateral circulation and

Figure 3 T1-weighted axial image after gadolinium administration shows the rim enhancement of the mass-lesion, as well as some central enhancement.

if sufficient to perform a parent vessel occlusion. On the follow-up angiogram six months later a spontaneous occlusion of the left pericallosal artery was demonstrated (figure 8, 9).

A follow-up angiogram 12 months later showed no changes.

Discussion

Spontaneous dissecting aneurysms in the anterior cerebral artery are extremely rare, especially in young patients. Mori et Al presented in a review 27 patients with dissecting aneurysms of the anterior cerebral artery with a mean age of 45 years, the three youngest being five, 27 and 37 years¹. Patients in a second review on dissecting anterior cerebral artery aneurysms had a mean age of 47 years, the youngest patient being six years². An older publication on spontaneous dissecting aneurysms in children and adolescence described only one patient with an anterior cerebral artery dissection³. The two recent review studies showed a strong male predominance 92% and 75%^{1,2}. In a recent review of fusiform aneurysms of the middle cerebral artery Day et Al postulated that these aneurysms develop as a result of an arterial dissection⁴. These dissect-

Figure 4 Thick-slice T1-weighted sagittal image depicts the relation between the mass lesion and the pericallosal artery, and it shows the lesion enhancement.

ing aneurysms are found in younger patients than the aneurysms in the posterior circulation. In many instances the etiology of dissecting aneurysms is unclear, however many associations have been described such as, hypertension, trauma^{1,2}, congenital vascular variation⁵, fibro-muscular dysplasia, alpha-antitrypsin deficiency⁶, and genetic causes⁷. The male predominance led Koyama too the hypothesis that intimal fibroelastic abnormalities related to sex hormones may play a role in the etiology of

Figure 5 Diffusion weighted axial images shows the infarcts in the left anterior territory.

Figure 6 Maximum intensity projection of a contrast enhanced MRA depicts the anatomic variation of the proximal A2, especially on the affected left site. The redundancy of the left pericallosal artery is clearly visible.

Figure 7 A) Left lateral projection after internal carotid injection. In the arterial phase the redundancy of the anterior carotid artery is illustrated by the two loops (A,B). The vessel is widened as a whole with a focal dilatation on the level of the genu of the corpus callosum, with slow filling of an aneurysm (B). The slow flow is visible in the venous phase where there is still enhancement of the left distal anterior cerebral artery (C).

dissecting aneurysms⁸. A common pathway in all dissecting aneurysms may be a fragmentation of the internal elastic lamina^{9,10}.

The natural history of dissecting aneurysms of the anterior cerebral artery is different from posterior dissecting aneurysms. In contrast to dissecting aneurysms of the posterior circulation, dissecting aneurysms of the anterior cerebral artery often present with thrombo-embolic complications. This may be due to the site of

dissection, in the vertebrobasilar system dissection occurs in 50% of cases between the media and adventitia, this has a higher chance of rupture to the subarachnoid space¹¹. Anterior circulation dissection is probably located between the elastica and media, and this may lead to thrombo-embolic complications⁴.

Because the dissection does often not result in a subarachnoid haemorrhage the initial dissection remains clinically silent unless it is associated with ischemia from luminal stenosis. Presumably many dissecting aneurysms resolve spontaneously. However, the vessel wall damage may lead to an expanding lesion resulting in a serpentine shape with intraluminal thrombosis. This will decrease the risk of subarachnoid haemorrhage, but increases the risk of thrombo-embolic complications. If the lesion increases in size it may lead to local mass effect and secondary ischemia and edema. This sequence of events was also seen in the presented case.

If we consider the etiology of the aneurysm in the presented case it is very unlikely that an acquired lesion or environmental factors play a significant role in aneurysm formation, because the patient is young and has no known risk factors. The prominence of the orbital frontal and frontal polar distribution, even more prominent on the left, and the absence of the callosomarginal artery may reflect an abnormal development resulting in an increased vulnerability. This hypothesis is supported by the association between vertebrobasilar junction fenestration and dissecting aneurysms⁵. On the other hand, the anatomic disposition in this patient is generally considered a normal variation¹².

Treatment options for dissecting aneurysms are limited. In a dissecting aneurysm with a neck, coiling may delay growths of the lesion and reduce the water hammer effect. If collateral supply is sufficient parent vessel occlusion can be an option, and if collateral supply is insufficient a vascular bypass may be used. In acute thrombo-embolic complications (intra-arterial) thrombolysis can be considered. However conservative treatment may also lead to a good outcome. In a review of 17 patients with dissecting aneurysm of the anterior cerebral artery presenting with an ischemic event, 11 patients were treated conservatively with a good outcome in ten patients and one patient was moderately disabled¹. A second review with some overlap in patients reported 20 patients with a dissecting aneurysm of the anterior cerebral artery presenting with ischemia. Of these patients, nine were treated conservatively with a good outcome in seven and a moderated outcome in two patients².

There is a consensus that more aggressive surgical treatment with arterial reconstruction may be appropriate in the patients presenting with a subarachnoid haemorrhage due to a dissecting aneurysm². However there is no consensus on the treatment of dissecting aneurysm presenting with thrombo-embolic complications. There seems to be a difference between the outcome of conservatively treated dissecting aneurysm of the medial cerebral artery and anterior cerebral artery. In a review of 13 medial cerebral artery dissecting aneurysms presenting with ischemia and treated conservatively, only one patient had a good outcome and five patients died². This may be due to the often proximal location of the aneurysm in the medial cerebral artery trunk.

Figure 8 Six months after initial presentation. Left lateral projection after left internal carotid injection. The pericallosal artery is occluded; only the fronto-polar branch of the left anterior cerebral artery is visible as well as media branches.

However more information is needed to determine an optimal treatment for dissecting aneurysms and anterior cerebral artery dissecting aneurysms in particular.

Figure 9 Six months after initial presentation. AP projection after right internal carotid injection, arterial phase. No crossover of the right anterior cerebral artery.

In the presented case only two treatment options were possible, conservative treatment with or without intra-arterial thrombolysis or anticoagulant therapy, and parent vessel occlusion. Because of the delayed and sub-acute presentation, thrombolysis was not considered appropriate. Parent vessel occlusion was considered but spontaneous occlusion of the left anterior cerebral artery distal to the fronto-polar artery occurred within a few months after initial presentation.

Conclusions

Dissecting aneurysms can be a devastating disorder and difficult to manage, however in the anterior cerebral artery conservative treatment has a good outcome in many patients. It is very unlikely that all dissecting aneurysms have the same etiology and natural history and without this knowledge it remains difficult to decide which treatment is optimal for a given patient.

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