Dissecting anterior inferior cerebellar artery aneurysm treated by endovascular route

Report of three cases and review

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Among the major arteries of the posterior fossa, the anterior inferior cerebellar artery (AICA) is the least likely to harbor an aneurysm, being a very rare pathology. Only two AICA aneurysms were found in 7,933 lesions¹. In the presence of a high flow lesion, aneurysms tend to occur more commonly in the feeding arteries. These flow-related aneurysms in the posterior fossa can be found mainly associated with arteriovenous malformations (AVM) or hemangioblastomas, which represents the majority of AICA aneurysms reported²,³. For eight years, 1,450 aneurysms were seen in a single-center, with only two AICA aneurysms, both flow-related (AVM)³.

Aside from flow-related aneurysms, almost all distal AICA aneurysms are dissecting aneurysms, usually presenting in otherwise healthy adults. Again the AICA is the least likely to present an isolated dissection among the major arteries. We report three cases of “spontaneous” AICA dissection aneurysms.

CASES

Case 1

A 47yo woman had a sudden headache with vomiting, followed by loss of consciousness. A CT showed diffuse posterior fossa subarachnoid hemorrhage. The angiography, that was immediately performed, revealed a distal right AICA aneurysm and a three dimensional reconstruction was done. The ipsilateral posterior inferior and the superior cerebellar arteries were present and with a very good diameter. Dissected AICA occlusion was decided upon and a microcatheter (ultraflow, ev3) was positioned just near do the diseased segment. A mixture of 33% cyanoacrylate and lipiodol was prepared and injected under subtracted fluoroscopy. The final control shows complete occlusion of the aneurysm and parent vessel.

Case 2

The second case is a 16-year-old female with complains of sudden headache and left partial facial palsy. The neurological exam revealed an important meningismus. The CT scan demonstrated a subarachnoid hemorrhage in the left pontine angle cistern and in the fourth ventricle. During angiography a distal left AICA fusiform aneurysm was demonstrated, involving the whole circumference of the vessel wall. Observe the late stagnation of contrast inside the aneurysm, suggesting the dissection etiology. A microcatheter was advanced just before the aneurysm and glue at 33% was injected (cyanoacrylate and lipiodol). The final angiogram confirms complete resolution. The partial left facial palsy had an almost complete recovery after 6 months.
Case 3
The third case is a 29-year-old man with two episodes of sudden headache within a week and no other clinical signs or symptoms associated. The first CT scan was normal and the second CT after the second episode showed a Fisher IV subarachnoid hemorrhage. An angiography was performed and a right distal AICA fusiform aneurysm was diagnosed. Again, observe the late stagnation of contrast inside the aneurysm and the diameter difference of the artery on the dissected segment. Once more, a microcatheter was advanced until the diseased portion of the artery, occluding the aneurysm, proximal and distal dissected segment with glue. The patient developed hydrocephalus and was shunted on the same day. The clinical follow-up was uneventful.

**DISCUSSION**
Isolated dissection of an intracranial branch vessel is rare, but through improvements in imaging techniques, especially MRI and Digital Subtraction Angiography (DSA), they are being diagnosed with increasing frequency. When intracranial dissection occurs, subarachnoid hemorrhage (SAH) or stroke can follow. The structural differences between systemic and intracranial arteries are known. Two planes of dissections are established. In patients with SAH, the plane of dissection is usually between the media and adventitia. The other plane is between the internal elastic lamina and media, resulting in occlusion of the vessel without adventitial disruption, leading to an ischemic event. The lack of the external elastic lamina and the thinner adventitia of the intracranial arteries may predispose to adventitia disruption during dissection external to the media, with consequent SAH and pseudoaneurysm formation.

Patients with distal AICA dissecting aneurysms may present symptoms and signs of typical SAH, with sudden severe headache, meningismus, nausea, vomiting, photophobia, and/or coma. More localizing presentations may be seen, especially in larger aneurysms with mass effect, tinnitus, hearing loss, vertigo, gait ataxia, diplopia, facial paresis and lower cranial nerve palsies. However, a SAH presentation is more common and was observed in 88% of patients with intracranial artery dissections in the posterior circulation. It can reflect a more intensive investigation when facing SAH, especially the role of DSA in which the features of dissection are well described.

Surgical treatment of distal cerebellar arteries dissecting aneurysms depends on their location but is considered to be difficult and associated with high morbidity and mortality rates. To spare the related artery is possible when there is a favorable neck and the aneurysm is not fusiform. Although surgery for distal cerebellar artery aneurysms is often difficult and with substantial morbidity, endovascular parent vessel occlusion is technically easier. A great number of these aneurysms is located on a distal branch and thereby limiting the area of possible induced infarction. In cases of insufficient collateral circulation, the area of infarction usually remains relatively restricted.

In these reported cases, the surgical treatment would consist in a parent artery occlusion, which can also be done by endovascular route. Eckard et al described nine cases in which the occlusion of a parent vessel with peripheral aneurysm was obtained with low complication rates. The potential risk is represented by distal ischemic damages in the parent vessel territory. In the posterior fossa, a wide variation in arterial distribution exists. Specific areas of the brain stem and cerebellum cannot always be predictably allotted to a particular cerebellar artery. The effects of occlusion of a cerebellar artery range from clinical silence to death, as a result of infarction of portions of brain stem or cerebellum.

Prediction of deficits have been tried with Amytal testing, but it does not seem to be helpful because collateral circulation is generally efficient in this cerebellar territory, preventing significant infarction. Furthermore, in our case, the treatment remains the same even if the test is not tolerated.

Excellent results have been reported in most recent studies evaluating the efficacy of parent vessel occlusion with coils or glue in the treatment of peripheral aneurysms. However, even if coils can be positioned, the diameter of the vessel and the fragility of the wall make them unsuitable for repeated mechanical manoeuvres and periprocedural reruptures may occur. Liquid embolic agents are commonly used for endovascular treatment of malformations. Cyanoacrylate, the most common used, polymerises after contact with blood and becomes solid, occluding the vessel completely and permanently. The use of liquid embolics for occlusion of saccular cerebral aneurysm may difficult the prevention of the agent migration to the parent artery, although, in dissecting fusiform aneurysm, complete occlusion of the dissected artery can be achieved with proximal glue injection, filling the whole dissected segment.

In conclusion, peripheral AICA ruptured dissecting aneurysm is a very rare disease and little information is available regarding its management. Parent artery occlusion may be proposed as a therapeutic alternative. When performing the occlusion by superselective embolization an excellent anatomic result and clinical outcome can be achieved.

**REFERENCES**