Dissecting Aneurysm of the Basilar Arterial Trunk Presenting with Pontine Infarction

Coil Obliteration of the Dissecting Aneurysm Including the Diseased Basilar Arterial Segment

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Summary

Dissecting basilar aneurysms have rarely been reported but are associated with high morbidity and mortality. Therefore, controversy exists as to the proper management of such lesions because their natural course is not well understood. We describe a 50-year-old man with a dissecting aneurysm involving the lower basilar trunk who presented with pontine infarction corresponding to the aneurysmal sac location. We obliterated the dissecting basilar aneurysm by coil embolization of the aneurysmal sac as well as the diseased segment of the basilar trunk after confirmation of collateral filling of the basilar artery through the posterior communicating artery. The patient recovered without any procedural complication. Eight month follow-up revealed complete disappearance of the aneurysm without symptom recurrence together with preservation of collateral flow in the distal basilar artery. Obliteration of the parent artery as well as the aneurysmal sac with coils could be considered in a lower basilar aneurysm of a dissecting nature.

Introduction

Dissecting aneurysms of the basilar artery (BA) not related to the vertebral artery (VA)

are rare. The natural history of dissecting basilar aneurysm is poorly understood although a morbid outcome has often been reported in contrast to some cases with a benign course, suggesting a less severe outcome of the dissecting aneurysm [1]. We report a basilar dissecting aneurysm involving the lower BA, treated with detachable coil embolization of the aneurysm as well as the diseased segment of the BA.

Case Report

A 50-year-old man presented with the right extremity weakness and dysarthria. He had experienced a dull headache for one month and it became aggravated at the time of admission. MRI performed upon admission, revealed a left pontine infarction (figure 1A).

Cerebral angiography revealed a dissecting pseudoaneurysm in the BA (figure 1B). We confirmed the collateral filling of the distal BA segments through the posterior communicating artery (PCoA) (figure 1C). The size of the aneurysm was 8 mm in the vertical, 5 mm in the transverse, and 4 mm in the anteroposterior dimension. The aneurysmal neck was almost the same as the vertical aneurysm size. Because of the dissecting nature of the aneurysm, we decided to obliterate the lesion to prevent pro-

Figure 1 A 50-year-old man presented with right extremity weakness. A) T2-weighted MR image shows a left pontine infarction. B) Anteroposterior view of the right vertebral arteriogram shows a dissecting aneurysm in the lower basilar trunk. The aneurysmal sac was not separate from the BA. Note faint filling of the BA distal to the aneurysm. C) The right internal carotid arteriogram shows a good filling of both posterior cerebral arteries and the left anteroinferior cerebellar artery. D) The right vertebral arteriogram reveals a broad neck as a nature of dissecting aneurysm involving the lower main trunk of the BA. E) Both VAs were patent after occlusion of the BA including the aneurysmal sac. The right internal carotid arteriogram (F) and the right vertebral arteriogram (G) obtained eleven days later show the same features of patent distal BA including the left anteroinferior cerebellar artery as in (C) and a flow dependent size decrease of the right VA beyond the right posteroinferior cerebellar artery. Eight month follow-up angiogram did not reveal any changes in the completely occluded aneurysm and diseased segment (not shown). The patient did not have any symptomatic recurrence during the eleven month follow-up period with progressive improvement of his symptoms.

gression of the pathology. Under neuroleptic anesthesia, a 5-F guiding catheter was placed in the left VA. After a microcatheter was advanced into the sac, a 5-mm diameter GDC (Boston Scientific, Fremont, Calif) was introduced as a frame. The aneurysmal sac was then gently packed with seven GDCs which were 4mm, 3mm, 2mm in diameter. Gradual occlusion of the lower BA occurred due to packing coils into the aneurysm (figure 1E). Because preservation of the BA was not possible and a

proper stent was not available at the time of the procedure, we decided to occlude the BA which had sufficient collateral filling via the PCoA (figure 1F). No significant complications occurred during this procedure. Heparin was infused in the amount of 5000 units at the beginning of the procedure and an additional 1000 unit infusion was given under activated clotting time monitored hourly. Follow-up angiogram obtained 11 days later confirmed complete exclusion of the aneurysm as well as the

dissected BA with a good patency of the upper and mid basilar trunk through the PCoA (figure 1G).

The patient's clinical condition improved after the procedure and he was discharged without further additional neurological deficit. Eight month follow-up angiogram revealed no recurrence of the aneurysm. The patient has only a mild dysarthria and the right upper extremity weakness without any further symptom aggravation on eleven month follow-up.

Discussion

Management of BA dissecting aneurysms is both controversial and challenging. The three options available to these patients are conservative management, surgical treatment and neurointervention. Occasionally, spontaneous resolution is observed in occlusive or stenotic dissections¹. There are three reported cases of mid-basilar dissecting aneurysms treated by endovascular occlusion of VA²⁻⁴. In our case, we

chose to perform coil embolization of the lower basilar dissecting aneurysm because serial angiography had demonstrated a sustained dissecting aneurysmal sac which corresponded to the patient's symptom.

In addition to the good patency of the collateral pathway as demonstrated in our case through the PCoA into the distal BA, concern related to BA occlusion is the possibility of perforator occlusion arising from the BA. The caudal group of basilar perforators originates in the dorsal surface between the vertebrobasilar junction and the anteroinferior cerebellar artery. One to four vessels which are identified in this region, descend along the basilar sulcus, and enter the foramen caecum in the medulla oblongata. Occasionally, as one or more of these vessels arise from the pontomedullary artery of the posteroinferior cerebellar artery, collateral flow develops when an obstructive lesion has occurred in this area⁵.

The rationale regarding proximal occlusion of the BA is supported by this possibility of collateral flow. There is currently no ideal or definitive therapy for basilar dissecting aneurysm. Therefore, clinical and radiological follow-up should be required after any intervention, and the effect of each type of clinical management should be further evaluated in larger patient study groups.

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