

Spontaneous Dissecting Aneurysm of the Anterior Cerebral Artery

Myoung Soo Kim, M.D., Chae Heuck Lee, M.D., Seung-Joon Lee, M.D., Jong Joo Rhee, M.D.

Department of Neurosurgery, Seoul Paik Hospital, Inje University College of Medicine, Seoul, Korea

Spontaneous dissection of the anterior cerebral artery is an unusual cause of subarachnoid hemorrhage. We present a case of a dissecting aneurysm of the anterior cerebral artery presenting with subarachnoid hemorrhage. A 51-year-old woman presented to our hospital with severe headache. Neurological examination demonstrated neck stiffness, decreased visual acuity of the left eye, and left ankle weakness. Computed tomographic scans showed subarachnoid hemorrhage. The initial cerebral angiogram demonstrated a slightly narrowed caliber and mild poststenotic dilatation of the right A1 segment. A second cerebral angiogram 14 days later revealed no change in the focal narrowing of the proximal A1 segment but marked progression of the dilatation of the distal A1 segment. Right pterional craniotomy was performed. A sausage-like dilation of the right A1 segment was found with no definite mural hematoma. This abnormal right A1 segment was wrapped with a Sundt clip. A postoperative computed tomographic scan revealed infarction of the right head of the caudate nucleus and the anterior limb of the right internal capsule. If a dissecting aneurysm is suspected, serial angiographic studies should be performed because of the possibility of dynamic changes over a short period.

KEY WORDS : Anterior cerebral artery · Dissecting aneurysm.

Introduction

Dissecting aneurysms of the anterior circulation remain rare but are being increasingly recognized as an important cause of cerebral infarction in younger patients and of subarachnoid hemorrhages¹². In Korea, there have been only two published reports of dissecting aneurysms of the anterior cerebral artery⁷. The risk of rebleeding from cerebral dissections associated with subarachnoid hemorrhage is high¹². We present a case of a dissecting aneurysm of the anterior cerebral artery presenting with subarachnoid hemorrhage. In addition, we discuss the diagnosis and treatment of dissecting aneurysm of the anterior cerebral artery presenting with subarachnoid hemorrhage.

Case Report

A 51-year-old woman presented to her local hospital after experiencing a sudden onset headache about one day earlier. She was noted to be hypertensive and confused. She

then presented a seizure attack. A computed tomographic scan at the local hospital showed subarachnoid hemorrhage throughout the basal cistern, interhemispheric fissure, and Sylvian fissure (Fig. 1). She was transferred to our hospital.

Physical examination on admission disclosed no abnormality except acute illness. Neurological examination demonstrated neck stiffness, decreased visual acuity of the left eye, and left ankle weakness. Eye examination showed multiple subretinal hemorrhages in the left eye.

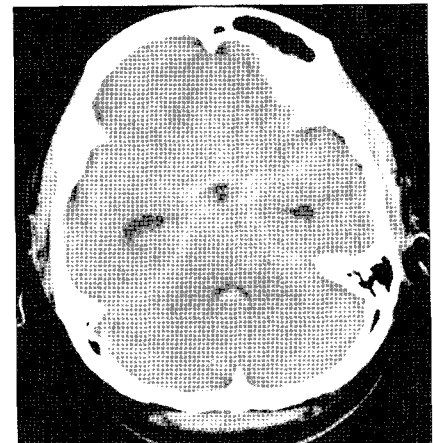


Fig. 1. Noncontrast computed tomographic scan demonstrating subarachnoid hemorrhage associated with dilation of the temporal horns of the lateral ventricle.

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• Address for reprints : Chae Heuck Lee, M.D., Department of Neurosurgery, Seoul Paik Hospital, Inje University College of Medicine, 85 Jeo-dong 2-ga, Jung-gu, Seoul 100-032, Korea Tel : +82-2-2270-0032, Fax : +82-2-2270-0573, E-mail : chleens@hotmail.com

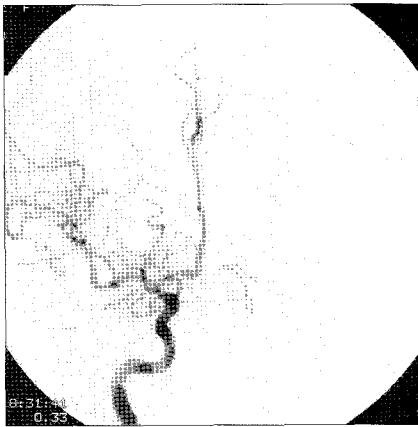


Fig. 2. Anteroposterior projection of the right internal carotid artery showing focal stenosis and mild poststenotic dilation of the right A1 segment.

The initial cerebral angiogram demonstrated a slightly narrowed caliber and mild poststenotic dilation of the right A1 segment of the anterior cerebral artery (Fig. 2). These findings were highly suspicious of an aneurysm or dissection of the right A1 segment although not diagnostic. Brain magnetic resonance imaging performed nine days later showed no abnormality except for a small residual subarachnoid hemorrhage. A second cerebral angiogram was arranged for 14 days later because of the highly suspicious of an underlying abnormality. This second-look angiogram revealed no change in a focal narrowing of the proximal A1 segment but marked progression of the dilation of the distal A1 segment (Fig. 3).

The patient was taken for operation 18 days after the subarachnoid hemorrhage. Right pterional craniotomy was performed. Small old subarachnoid hemorrhage mixed cerebrospinal fluid was found after dura incision. After exposure of the right internal carotid artery and optic nerve, the right A1 and A2 and left A1 segments were identified but no berry aneurysm was found. Instead, a sausage-like dilation of the right A1 segment was found, which appeared to have no definite mural hematoma. This abnormal right A1 segment was wrapped with a Sundt clip (Codman & Shurtleff, Inc., Randolph, MA). Micro-doppler examination demonstrated good flow in the right A1, right A2 and left A1 segments.

A postoperative computed tomographic scan revealed infarction of the head of the right caudate nucleus and the ant-

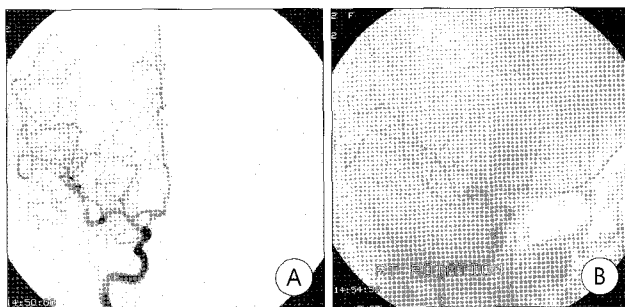


Fig. 3. Right internal carotid angiograms two weeks after the first angiogram, demonstrating no change in the stenosis and a marked increase in the poststenotic dilation of the right A1 segment (A : anteroposterior view, B : oblique view).

erior limb of the right internal capsule (Fig. 4). She slowly recovered from the anesthesia and showed no further neurological deficit. A cerebral angiogram 30 days after the operation revealed complete obliteration of the right A1 aneurysm and right distal A1 segment. Moreover, good collateral flow in

the right A2 segment through the anterior communicating artery was shown (Fig. 5). She was discharged two months after the aneurysm operation, with mild weakness of the left ankle and decreased visual acuity of the left eye (visual acuity of right eye : 1.0; visual acuity of left eye : hand motion at 30cm).

Two months after the aneurysm operation, she underwent vitrectomy of the left eye for Terson's syndrome. Two years and five months after the aneurysm operation, she demonstrated no left ankle weakness and improved left visual acuity (visual acuity of right eye : 1.0; visual acuity of left eye : 0.1).

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Discussion

Diagnosis of anterior cerebral artery dissecting aneurysm

Angiography may detect dissecting aneurysms as narrowing and dilation, pooling, slow filling, or a filling defect. These findings are not considered particularly specific, as they are also seen in atherosclerotic vascular diseases. The only pattern that can be reliably regarded as a diagnostic sign is the double

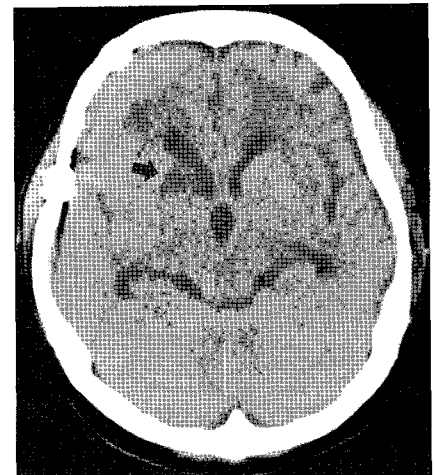


Fig. 4. Non-contrast computed tomographic scan after surgical clipping of the right A1 segment dissecting aneurysm, demonstrating an infarct in the head of the right caudate nucleus (arrow).

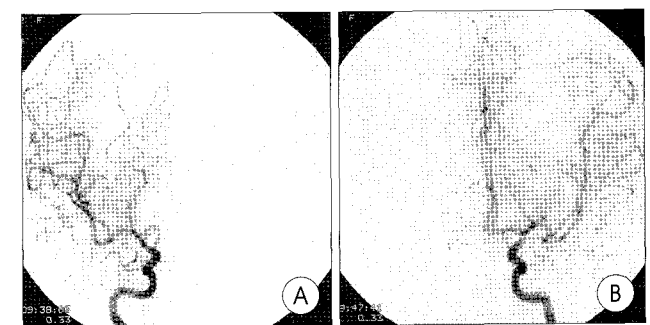


Fig. 5. Postoperative internal carotid angiograms showing partial filling of the right A1 segment from the right-sided injection (A) and both A2 segments filling from the left-sided injection (B).

lumen, in which both true and false lumens are seen⁸⁾. However, angiography does not always allow diagnosis of a dissecting aneurysm as dissections involving small-caliber vessels may be overlooked. Surgical inspection is the only method to confirm the dissecting aneurysm in some cases¹⁴⁾.

No aneurysm is angiographically detected in 15 to 20% of spontaneous subarachnoid hemorrhage patients¹⁶⁾. In our case, aneurysm was not detected in the first angiogram. The only angiographic finding in the first angiogram was a smooth stenosis and a poststenotic dilation. The second angiogram, performed after two weeks, demonstrated a markedly progressive poststenotic dilation as a dramatic change. A dissecting aneurysm should always be considered if the angiogram detected no aneurysm. Rinkel states that subarachnoid hemorrhages without a detectable aneurysm in patients with diffuse or anteriorly located blood on computed tomographic scan are at risk of rebleeding and have a poor prognosis¹⁶⁾. In addition, Rinkel states that the origins of these subarachnoid hemorrhages might be arterial dissection, dural arteriovenous fistulae, mycotic aneurysms, trauma, bleeding disorders, substance abuse, or a cervical origin. In the management of subarachnoid hemorrhage of unknown origin, surgical inspection is important for diagnosis and in prevention of rebleeding.

A definitive diagnosis of a dissecting aneurysm is sometimes difficult to make because imaging may reveal occlusive lesions. Other well-known angiographic findings such as the pearl and string sign and the rosette sign are also found in atherosclerotic disease. A double lumen with opacification of both true and false lumens may be a specific diagnostic sign; however, it appears on angiograms only occasionally^{2,3,9,11,17)}. Angiographic signs may be understood in terms of the status of the thrombosis in the pseudolumen; thus, a double lumen could result from minimal thrombosis, the pearl and string sign could result from partial thrombosis, and subtle stenotic or occlusive lesions could result from early progression of thrombosis. Although dissecting aneurysms often demonstrate only subtle abnormalities in the acute stage, early thrombosis might not necessarily indicate a dissected pseudolumen¹¹⁾. More findings that are common are dilation, narrowing, pooling of contrast medium, and occlusion of the anterior cerebral artery⁶⁾. Cerebral vasospasm due to subarachnoid hemorrhage occasionally has the appearance of a string sign. Cerebral vasospasm commonly occurs in multiple vessels and is scarcely seen at the acute stage. Differentiation from the stenotic or occlusive lesions in incidental atherosclerotic stenosis may be important. Another definitive diagnostic feature of arterial dissection is intramural hematoma^{5,11,15)}. Magnetic resonance imaging (MRI) is valuable when combined with angiography because MRI can directly demonstrate the intramural hematoma^{5,11,15)}. MRI can allow successful diagnosis

of dissecting aneurysms as the hemorrhagic sources among subarachnoid hemorrhages of unverified etiology and MRI discriminates them from atherosclerotic stenosis¹¹⁾. However, in this case, we could not find any mural hematoma on MRI. It seems probable that angiographically dynamic changes represent a characteristic of this disease and distinguish it from occlusive diseases due to other etiologies that may exhibit a similar appearance on angiography⁶⁾. Thus, serial angiography is essential for diagnosing and following up this disease. In our case, the first angiography showed only stenosis and subtle dilation of the anterior cerebral artery. The second angiography, after two weeks, demonstrated progression of the poststenotic dilation of the anterior cerebral artery.

Treatment of anterior cerebral artery dissecting aneurysm

The mechanism responsible for bleeding is thought to be transmural perforation due to the dissection. Intracranial dissecting aneurysms can be classified into the two types based on clinicopathological findings¹⁹⁾. The primary mechanism by which a cerebral dissecting aneurysm is created is the sudden disruption of the internal elastic lamina. The plane of dissection extends through the media. The majority of aneurysms have one entrance into the pseudolumen (entry-only type). This type is associated with an unstable clinical course. Some cerebral artery dissecting aneurysms have both an entrance and exit (entry-exit type). This type of aneurysm occasionally contains a constant flow of blood through the pseudolumen and is clinically more stable than entry-only aneurysms¹⁰⁾.

Early surgery may be justified to prevent rebleeding. If a diagnosis of dissecting aneurysm is highly suspected, trapping of the aneurysm or proximal ligation of the artery with or without extracranial-intracranial bypass surgery or wrapping of the dissecting aneurysm would be the optimal surgical treatment for prevention of rebleeding, although the best surgical procedure is still being debated^{11,17)}. A substantial proportion of dissecting aneurysms showing only subtle angiographic stenotic or occlusive lesions in the acute stage was noted. There was a high mortality rate due to subsequent rupture of such stenotic or occlusive lesions. Compared with the devastating mortality caused by the subsequent ruptures, the extent of surgical morbidity was minor. Surgical intervention may therefore be justified when the following neuroradiological findings are present: 1) angiographic stenotic or occlusive lesions; 2) distribution of subarachnoid hemorrhage on computed tomography compatible with the location of the angiographic stenotic or occlusive lesions; and 3) intramural hematoma on MRI in the same region as the angiographic stenotic or occlusive lesions¹¹⁾.

Some reports have advocated surgical intervention to prevent delayed deterioration secondary to bleeding^{4,13}, while others favor conservative treatment because arterial dissection is likely to heal by itself and there is a risk of brain injury associated with surgical manipulations, especially in the acute stage^{1,2}. Direct exploration is diagnostic and is recommended for patients with subarachnoid hemorrhage as subsequent absorption and resolution of the clots could result in a fatal rupture. As the cerebral arteries lack an external elastic lamina and have only a thin adventitia¹⁸, subintimal hematomas could rupture into the subadventitial plane and subarachnoid space, especially in older patients with more advanced medial defects. Furthermore, if progressive dissection or embolism by the clots occludes the distal branches, operative procedures would become necessary⁴. Trapping is the most effective procedure, and concurrent bypass surgery should be considered in cases where the collateral circulation is thought to be insufficient. Therefore, surgeon must carefully determine the surgical method according to the hemodynamic variations in each patient. Wrapping may be suitable in some cases¹³.

Conclusion

We believe that in patients with subarachnoid hemorrhage without apparent causative complications, the presence of a dissecting aneurysm should be considered even in the absence of apparent abnormalities on the initial angiogram. If a dissecting aneurysm is suspected, diagnostic examinations and serial angiographic studies should be performed immediately because of the possibility of dynamic changes over a short period.

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