Spontaneous Anterior Cerebral Artery Dissection Presenting with Simultaneous Subarachnoid Hemorrhage and Cerebral Infarction in a Patient with Multiple Extracranial Arterial Dissections

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Simultaneous subarachnoid hemorrhage and infarction is a quite rare presentation in a patient with a spontaneous dissecting aneurysm of the anterior cerebral artery. Identifying relevant radiographic features and serial angiographic surveillance as well as mode of clinical manifestation, either hemorrhage or infarction, could sufficiently determine appropriate treatment. Enlargement of ruptured aneurysm and progressing arterial stenosis around the aneurysm indicates impending risk of subsequent stroke. In this setting, prompt treatment with stent-assisted endovascular embolization can be a reliable alternative to direct surgery. When multiple arterial dissections are coexistent, management strategy often became complicated. However, satisfactory clinical results can be obtained by acknowledging responsible arterial site with careful radiographic inspection and anti-platelet medication.

Key Words: Dissecting aneurysm · Subarachnoid hemorrhage · Cerebral infarction · Anterior cerebral artery · Endovascular embolization.

INTRODUCTION

Arterial dissection occurring at the vertebrobasilar system often presents as subarachnoid hemorrhage (SAH), while dissecting aneurysm at the anterior circulation tends to manifest infarction. Although the exact prevalence has not been reported, spontaneous dissection at the anterior cerebral artery (ACA) is infrequent, and its presentation as simultaneous occurrence of infarction and SAH is quite rare. Treatment of symptomatic ACA dissection should be directed to prevent further progression of the stroke on the basis of clinical presentation, either hemorrhage or infarction. Managements include conservative, endovascular, and direct surgical method. When multifocal extracranial dissections are coexistent with such symptomatic intracranial dissection, management strategies often became annoying and complicated. In this condition, treatment should be kept pace with serial angiographic and close clinical surveillance.

We describe a case of spontaneous ACA dissecting aneurism presenting with simultaneous SAH and infarction in a young adult with multiple extracranial dissections with emphasis on clinical manifestation and serial angiographic follow-ups.

CASE REPORT

This 37-year-old man with hypertension came to the Emergency room with complaints of sudden onset headache and right leg weakness after he stretched and yawned. Neurological examination showed alert mental state, frontal headache, and grade III weakness of the right leg. He had no remarkable past medical history, except nonspecific dizziness and headache since he was 25 years old. Head computed tomogram (CT) revealed scattered SAH at the left frontal convexities. A magnetic resonance (MR) images showed acute infarction at the rostrum of the left corpus callosum and double-lumen signal with high-signal hemorrhage (Fig. 1). Catheter angiogram showed multifocal dissections at the left carotid bulb and right vertebral artery with tiny aneurysm at the left proximal A2 (Fig. 2). His paresis seemed to be attributed...
Recently, intracranial dissection or dissecting aneurysm is increasingly recognized. It is attributed to heightened clinical awareness and subsequent identification of relevant clinical and radiological features. Nevertheless, various nonspecific manifestations often made prompt diagnosis and management difficult. Vertebrobasilar dissecting aneurysms generally present with SAH because of its elongated subarachnoid course, and are more frequently reported. Those confined in the anterior circulation are mainly involved in the supraclinoid carotid and middle cerebral arteries, and present with either hemorrhage or ischemia.

Isolated ACA territory infarction is very rare, representing 0.5-3% of all ischemic strokes, and angiographically proven ACA dissection is responsible for 47% of them. The ACA dissection is more difficult to identify than vertebrobasilar system because of narrower vessel calibers and more curved features. Therefore, if encountered with ACA territory infarction particularly in young patients without trauma or existing atherosclerotic vasculopathy, clinicians should reckon the possibility of dissection and warn the risk of bleeding or re-occlusion.

There are many radiographic clues suggesting a dissection, including “double-lumen”, “intimal flap”, “pear and string”, “string”, “tapered occlusion”, and “hyperintense intramural signal” on conventional angiogram, MRI, MRA, or CTA. Besides other features, dynamic change on serial angiography is the most culprit evidence of dissecting aneurysm. In our report, false lumen with hyperintense rim around the true lumen on MRI, and serial angiographic changes such as aneurysm enlargement and to the infarction. After 3 days, dual antiplatelet agents were prescribed to prevent further ischemia and his weakness was gradually improved.

He underwent two more CT angiograms during the next months. Final angiogram revealed enlargement of the left A2 aneurysm and progression of focal stenosis along the ACA (Fig. 3A). Endovascular intervention was performed under a general anesthesia by placing a 4.5×28 mm Enterprise stent (Cordis Neurovascular, Miami Lakes, FL, USA) through the A1 into the A2 segment, and deploying three Axium detachable helix coils (ev3 Inc., Irvine, CA, USA) into the aneurysm sac. Postoperative control angiogram revealed successful coil packing and flow preservation through the bilateral A2 segments (Fig. 3B). Six months postoperatively, angiogram showed neither growth of aneurysms nor recurrence of stenosis (Fig. 3C). He has no neurologic deficits and awaits another angiogram 12 months hereafter.

**DISCUSSION**

**Fig. 1.** Magnetic resonance images at admission show an acute ischemia of the left rostral corpus callosum in diffusion-weighted image (A) and apparent diffusion coefficient map (B). A fluid-attenuated inversion recovery image shows both infarction and hemorrhage of the left frontal convexity (C) and axial source image indicates hyperintense double lumen sign at the proximal anterior cerebral artery (white arrow) (D).

**Fig. 2.** Angiogram shows multifocal dissections at the left anterior cerebral artery (A), right vertebral artery (V3) (B) and right carotid bulb (C).

**Fig. 3.** After 4 months, angiogram shows superior elongation of the aneurysm sac and progressing stenosis along the anterior cerebral artery (ACA) (A). Postoperative angiogram shows complete filling of the aneurysm (B), and 6-month follow-up angiogram depicts no residual filling and straightening of the ACA due to a deployed stent (C).
progressive vessel narrowing the aneurysm are definite clues of
dissection. All these findings warrant immediate treatment oth-
er than close observation.

A dissection occurring between the internal elastica and the
media mainly presents as an ischemic stroke with occlusion of
the affected portion. In this circumstance, pushing the arterial
wall outward in radial fashion with stent that guarantee lumen
patency could be a primary treatment option. And, antiplatelet
medication with serial angiography should be considered as
strong additional measures for sub-intimal dissection. Another
subtype, sub-adventitial dissection between the media and
the adventitia usually presents as hemorrhage and leads to poor
prognosis than ischemic counterpart. In this subtype of
loosely woven vessel wall, treatment should be directed to seal
off the leakage to prevent further hemorrhage by occluding the
diseased vessel lumen harboring ruptured aneurysm. In this
case, simultaneous occurrence of SAH and infarction repres-
ents both sub-intimal and sub-adventitial dissection. This in-
dicates more severe and deeper dissection which potentially re-
results in recurrent event of hemorrhagic or ischemic stroke.

Extracranial arterial dissection is recognized as a cause of tran-
sient ischemic attack and primary or recurrent stroke. However,
clinical presentation is subtle and unnoticed in most cases. Be-
cause of considerable rate of spontaneous healing, there remains
controversy over optimum treatment. But, antiplatelet medica-
tions are current standard for care unless contraindicated or un-
stable. In this subtype of loosely woven vessel wall, treatment
should be directed to seal off the leakage to prevent further
hemorrhage by occluding the diseased vessel lumen harboring
ruptured aneurysm. In this case, simultaneous occurrence of SAH
and infarction represents both sub-intimal and sub-adventitial
dissection. This indicates more severe and deeper dissection
which potentially results in recurrent event of hemorrhagic or
ischemic stroke.

If hemorrhage is the first presentation, or angioarchitecture is
ever changing, antiplatelet medications would not be suffi-
cient. When such alteration is detected, proximal occlusion,
resection of lesion, or endovascular obliteration with or without
revascularization should be carefully selected. It should be deci-
ed after careful assessment of ruptured aneurysm, vessels prox-
imal and distal to the aneurysm, and status of collaterals. Endo-
vascular treatment can be a strong alternative when surgical
approach is not feasible due to inherent fragility in vessel wall.
Stent-assisted coiling could salvage proximal parent artery sim-
taneously obliterating aneurysm without compromising vessel.
If vascular territory infarction already occurred and chance of re-
covery is quite low, obliteration without revascularization also
might be considered. As previously reported, the outcome of
the ACA dissection patients is better than that of the other
ACA stroke patients after appropriate treatment.

CONCLUSION

An ACA dissecting aneurysm presenting with acute onset
SAH and infarction in a young adult patient should be regarded
as a high risk for subsequent stroke, albeit quite rarely. Although
radiographic findings are often uncertain for dissection, serial
angiographic surveillance is crucial to detect an on-going risk of
stroke. When changes in the angiographic architecture are iden-
tified, endovascular or surgical management with or without re-
vascularization should be meticulously considered.

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