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 antiplatelet medication.

Key Words: Dissecting aneurysm · Subarachnoid hemorrhage · Cerebral infarction · Anterior cerebral artery · Endovascular embolization.
rologic deficits and awaits another angiogram 12 months hereafter.

**DISCUSSION**

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 than ACA dissections. 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

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**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 other 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 poorer prognosis than ischemic counterpart[1,5,11,12]. 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.

Extracranial arterial dissection is recognized as a cause of transient ischemic attack and primary or recurrent stroke. However, clinical presentation is subtle and unnoticed in most cases. Because of considerable rate of spontaneous healing, there remains controversy over optimum treatment. But, antiplatelet medications are current standard for care unless contraindicated or unstable. In such alteration is detected, proximal occlusion, resection of lesion, or endovascular obliteration with or without revascularization should be meticulously considered.

If hemorrhage is the first presentation, or angioarchitecture is ever changing, antiplatelet medications would not be sufficient[1,3,5]. When such alteration is detected, proximal occlusion, resection of lesion, or endovascular obliteration with or without revascularization should be carefully selected. It should be decided after careful assessment of ruptured aneurysm, vessels proximal and distal to the aneurysm, and status of collaterals. Endovascular 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 simultaneously obliterating aneurysm without compromising vessel. If vascular territory infarction already occurred and chance of recovery 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[10].

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 identified, endovascular or surgical management with or without revascularization should be meticulously considered.

References


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