www.jkns.or.kr http://dx.doi.org/10.3340/jkns.2013.54.3.236
Print ISSN 2005-3711 On-line ISSN 1598-7876
J Korean Neurosurg Soc 54 : 236-238, 2013

Case Report

Rupture of *De Novo* Anterior Communicating Artery Aneurysm 8 Days after the Clipping of Ruptured Middle Cerebral Artery Aneurysm

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Rapidly developed *de novo* aneurysm is very rare. We present a rapidly developed and ruptured *de novo* anterior communicating aneurysm 8 days after the rupture of another aneurysm. This *de novo* aneurysm was not apparent in the initial 3-dimensional computed tomography and digital subtraction angiography. We reviewed the literature and discussed possible mechanisms for the development of this *de novo* aneurysm.

Key Words : *De novo* aneurysm · Subarachnoid hemorrhage · Aneurysm formation · Computed tomography angiography · Digital subtraction angiography.

INTRODUCTION

Since '*de novo*' aneurysm was first reported by Graf and Hamby[10] in 1964, well-documented case reports of *de novo* aneurysm within a short time interval are rare[1,18,19]. In general, the incidence of *de novo* aneurysms is uncertain and the time course of their development remains unclear. Especially, within several days' follow-up interval image studies, it is difficult to discern whether a *de novo* aneurysm is really new or was already present but unrecognized at the time of the first angiographic study.

We present a case of subarachnoid hemorrhage (SAH) caused by rupture of a middle cerebral artery M1 in 47-year-old man. Eight days later, he had an intraparenchymal hematoma caused by rupture of a '*de novo*' anterior communicating artery aneurysm. The anterior communicating artery aneurysm was not apparent in the initial 3-dimensional computed tomography (3D-CT) and conventional angiogram.

CASE REPORT

A 47-year-old man with subarachnoid hemorrhage in Hunt & Hess grade III was admitted via emergency room. The initial brain CT revealed a thick subarachnoid blood clot around basal cistern and left sylvian fissure (Fig. 1A). A 64-detector 3D-CT angiography showed a saccular aneurysm on the left M1 portion of middle cerebral artery and no additional aneurysms (Fig. 1B). Considering the distribution of the hemorrhage on CT scans and 3D-CT angiography, we concluded that the cause of the hemorrhage was rupture of the left M1 aneurysm. Immediately, we underwent pterional approach and aneurysmal clipping. On the microsurgical view, M1 aneurysm was surrounded with clots and we confirmed the obliteration of the aneurysm. The patient's mentality was improved to Hunt & Hess grade II. On the next day, right side hemiparesis was developed and the diffusion weighted magnetic resonance images showed ischemic change on the left posterior limb of the internal capsule (Fig. 1C). We performed digital subtraction angiography (DSA) which revealed mild vasospasm and performed intra-arterial nimodipine injections. There was no abnormal finding in the anterior communicating artery (Fig. 1D).

At eight days after the clipping, sudden deterioration of mentality appeared and the follow-up brain CT showed an intraparenchymal hematoma on the right frontal lobe (Fig. 2A). We performed the DSA again, which revealed severe vasospasm on the left M1 and A1 portion and the left anterior communicating artery aneurysm that was not found in the previous 3D CT an-

• Received : November 27, 2012 • Revised : August 28, 2013 • Accepted : September 15, 2013
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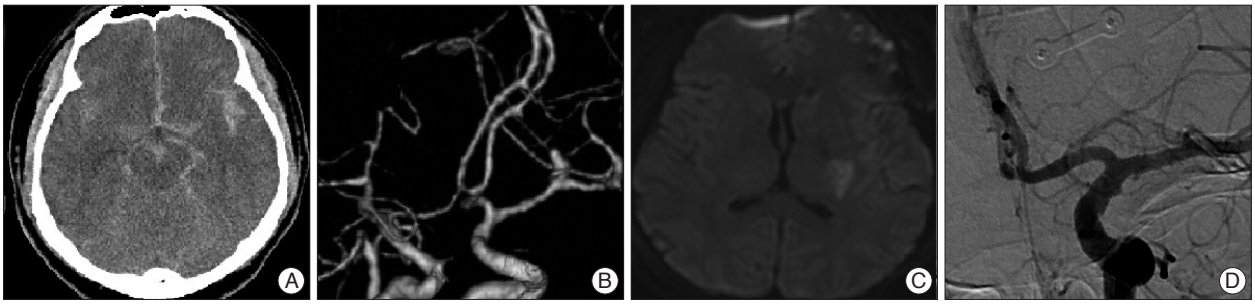

Fig. 1. A : Initial brain computed tomography (CT) showing subarachnoid hemorrhage in basal cistern and left sylvian fissure. B : A 64-detector 3-dimensional CT angiogram showing a saccular aneurysm on the left M1 portion of middle cerebral artery. There is no abnormal finding in anterior communicating artery. C : 1 day after clipping, diffusion weighted magnetic resonance image showing ischemic change of left posterior limb of internal capsule. D : 1 day after clipping, the trans-femoral catether angiography showing no abnormality in anterior communicating artery except mild vasospasm in peripheral arteries (not shown).

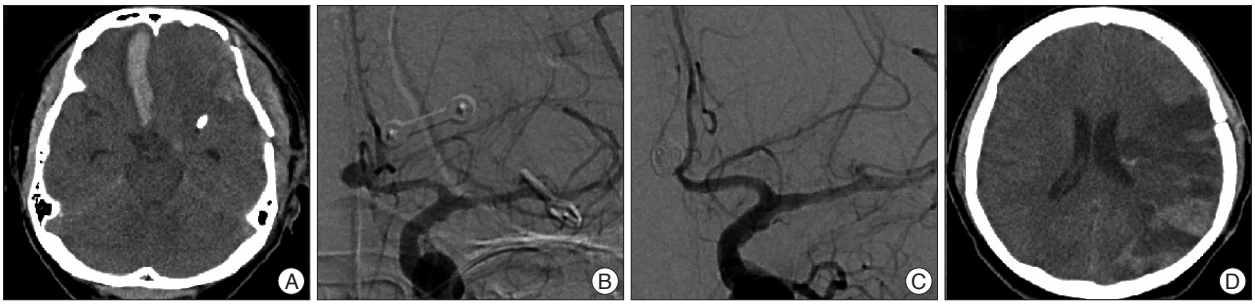

Fig. 2. A : Eight day after clipping, follow-up brain CT showing intraparenchymal hematoma in the frontal lobe. B : Eight days after clipping, follow-up trans-femoral catether angiography (TFCA) showing the anterior communicating artery aneurysm (4.9×4.9 mm) that was not existed in previous images with a massive general vasospasm. C : Simultaneously with TFCA, anterior communicating artery aneurysm was obscured by coil embolization and intra-arterial nimodipine injection was performed. D : Four weeks after the coil embolization, follow-up CT showing new developed cerebral infarction in the middle cerebral artery territory due to vasospasm and hemorraghic transformation.

giography and DSA (Fig. 2B). Simultaneously, coil-embolization was performed for the anterior communicating artery aneurysm with Target coils (4×10, 3×6, 2×4, and 2×2) (Boston scientific, Stryker) and it was obliterated completely on the final angiography. We injected intra-arterial nimodipine for the vasospasm (Fig. 2C). After the coil embolization, the patient was kept in confused mentality status. However, one month-follow up CT scan showed left cerebral hemisphere ischemia due to vasospasm and hemorraghic transformation (Fig. 2D). He was cooperative but was discharged with moderately severe disability due to right hemiplegia (mRS 4).

DISCUSSION

Fast-growing *de novo* aneurysm, reported within several days, with no evidence of another aneurysm is very rare. Most of *de novo* aneurysms were reported several years after the initial angiography[6,18,21,22]. However, Abe et al.[1] reported an unruptured *de novo* MCA aneurysm after 10 days, Schebesch et al.[19] reported a ruptured *de novo* basilar tip aneurysm after 44 days and Yasuhara et al.[23] reported a ruptured ophthalmic segment aneurysm after 47 days. We detected a *de novo* aneurysm within 8 days from the first CTA and one day after DSA. At first, we considered that we might have missed an anterior communicating artery aneurysm that was concealed from thick SAH or intra-aneurysmal thrombosis. However, considering the aneurysm size (4.9×4.9 mm), there was small amount of SAH around the anterior communicating artery on the initial CT. We concluded that the newly detected aneurysm was too large to be concealed by the small amount of SAH or intra-aneurysmal thrombosis. There was no definite abnormal finding in initial CTA. Furthermore, the day after, DSA showed no abnormal finding except mild vasospasm. Recently, the effectiveness of CTA in detecting aneurysms has been evaluated by several reports. Agid et al.[2] reported that sixty-four-detector CTA of the brain has been shown to be 98% sensitive and 100% specific for the detection of aneurysms in the setting of SAH. In their report, a small (1.7×2.0 mm) anterior communicating artery aneurysm was missed on CTA, which was identified on a retrospective review of the CTA after detection on DSA. Therefore, CTA could show all aneurysms even small one. Another recent study[17] reported that CTA resulted in a 0% false negative rate (sensitivity 100%, predictive value 100%) comparing with DSA in detecting aneurysms. Consequently, we conclude that this case was a *de novo* aneurysm or at least a rapidly growing aneurysm in a short time (8 days) frame for two reasons; 1) small amount of SAH around the anterior communicating artery and relatively large of aneurysm, 2) the accuracy of CTA & DSA for the diagnosis of aneurysm in recent studies.

In general, the pathophysiology and etiology of rapid develop-

ing *de novo* aneurysms is poorly understood. However, similar to patients with aneurysms in general, risk factors for the *de novo* aneurysms include hypertension, middle age, female, smoking and genetic predisposing factors-Marfan syndrome, fibromuscular dysplasia and Moyamoya disease[9,11,12,14-16]. A change in the hemodynamic environment caused by major vessel ligation[3], after stent placement[7], and after removal of arteriovenous malformation[5,20] may induce the *de novo* aneurysm by overloading some vascular territories. Moreover, severe vasospasm could have induced massive hemodynamic changes that finally resulted in the development of a new aneurysm[19]. The relation between the hemodynamic change and aneurysmal formation with growth is well established. Meng et al.[13] demonstrated that high wall shear stress initiate aneurysm formation. Also, Boussel et al.[4] showed that aneurysm growth occurs at the region of low wall shear stress. Either high or low wall shear stress may induce formation and growth of aneurysms. Recently, Doenitz et al.[8] reported a case study of a patient with a *de novo* basilar tip aneurysm that developed over 44 days and ruptured[19]. Their patient had severe vasospasm without other genetic predisposing factors. They analyzed flow parameters of the basilar artery before and after formation of the aneurysm with computational fluid dynamics. They found that low wall shear stress of the vessel corresponded to the site of aneurysm formation and growth, furthermore, impingement point and wall pressure had no clear relation. Based on this finding, they proposed a mechanism of genesis of fast-growing aneurysms. In our case, as with Doenitz's patient, the patient had severe vasospasm without any genetic predisposing factors. Therefore, we believe that the mechanism suggested by Doenitz et al. explains our patient's rapidly developing *de novo* aneurysm and rupture.

CONCLUSION

We present a rapidly developed and ruptured *de novo* anterior communicating aneurysm 8 days after the rupture of another aneurysm. That *de novo* aneurysm was not apparent in the initial 3D-CT and conventional angiogram.

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