INTRODUCTION

Spontaneous thrombosis seems to occur in natural course of unruptured large or giant aneurysm\(^3\). But it can occur even after a rupture of a relatively small aneurysm\(^2,6,10\). In the acute setting of subarachnoid hemorrhage (SAH), it is a situation that required caution because spontaneous thrombosis is likely not a true cure\(^10\). And the timing of its recanalization is unpredictable, and subsequent fatal rebleeding is uncontrollable. Although ruptured aneurysm at any location can be spontaneously thrombosed, reports on spontaneous disappearance of ruptured distal anterior cerebral artery (DACA) aneurysm are scarce\(^6,10\). Here, we describe an exceptional case of spontaneous thrombosis and recanalization of ruptured pericallosal artery aneurysm.

CASE REPORT

A female patient in her 40s, without underlying disease visited the emergency room with deteriorating consciousness. Computed tomography (CT) scan revealed massive intraventricular hematoma with acute hydrocephalus and frontal intracerebral hematoma (Fig. 1). The patient presented semicomatose mentality with Glasgow coma scale (GCS) 1/1/2, World Federation of Neurosurgical Society grade V on the initial assessment. CT angiography showed definite saccular aneurysm on pericallosal artery. But, cerebral angiography while attempting urgent coil embolization showed disappearance of the ruptured aneurysm along with thrombotic occlusion of parent artery. Then, the patient had been receiving conservative management in the intensive care unit. The CT was repeated on hospitalization day (HD) 7 and 14. Recanalization was detected on CT of HD 22. The ruptured aneurysm was obliterated with endovascular coiling on the HD 23. The aneurysm has been stable for 36 months. Careful surveillance for recanalization followed by delayed intervention will be crucial in the exceptional situations of a spontaneously disappearing aneurysm.

Keywords: Subarachnoid hemorrhage; Intracranial aneurysm; Angiography, Digital subtraction
postoperative months, functional state of the patient was GCS 4/3/6 and mRS score 4. Follow-up DSA at 36 months postoperatively revealed no recurrence other than the small residue of the aneurysm neck.

DISCUSSION

Several studies have suggested possible mechanisms of spontaneous thrombosis. Turbulent flow and subsequent endothelial damage appear to be a major cause of spontaneous thrombosis in
large or giant aneurysm). It is speculated that use of the antifibrinolytics, vasospasm, and elongated aneurysm shape with thick hematoma around the aneurysm may be the mechanism of spontaneous disappearance of small ruptured aneurysms. On DSA of our case, disappearance of the aneurysm is obviously due to thrombosis of the aneurysm and parent artery (Fig. 3). It clearly shows filling defects inside the lumen not the segmental narrowing as in vasospasm.

A blood blister-like aneurysm (BBA) can exhibit rapid morphological changes over time. Rarely, BBA can occur in locations other than the ICA, and it may have a saccular shape. However, the location of the denitite bifurcation site in our case does not match the typical BBA location of the nonbranching site. Although spontaneous thrombosis may occur in the clinical course of ruptured dissecting aneurysm, the dissecting feature of proximal narrowing with pseudoaneurysm formation could not be found in angiograms in our case.

Spetzler et al. described a possible relation between spontaneous thrombosis and the antifibrinolytics for preventing clot lysis induced rebleeding. In a systematic review, it was reported that antifibrinolytic treatment reduced rebleeding rate of ruptured aneurysm up to 40%. But it is not recommended as a routine practice because it does not improve mortality and clinical outcome. The situation was an exception, since we had no choice but to perform an EVD before aneurysm repair. Therefore Tranexamic acid (TXA) was inevitably used to reduce the possibility of rebleeding. A recent multicenter study reported that even with no statistical significance, procedural thrombosis was more common in the group of SAH patients with TXA treatment. Since we experienced this case, we have not used TXA in patients scheduled for urgent endovascular treatment.

Reports on spontaneous thrombosis of DACA aneurysms are scarce. Yoshikazu et al. reported findings similar to our case. In their report, ruptured DACA aneurysm was seen in CTA at 2.5 hours after rupture, but DSA after 8.5 hours from rupture failed to reveal aneurysm. They suggested an elongated shape with a narrow neck and thick hematoma compressing the aneurysm as a cause of spontaneous thrombosis. The CT finding of a large intracerebral hematoma around the aneurysm in our case was similar to their report.

The recanalization timing after spontaneous thrombosis of a ruptured small aneurysm is difficult to predict accurately. According to few previous reports, entire recanalization was detected in DSA on days 14-19 from the detection of spontaneous thrombosis. The time interval between follow-up imaging studies also differed between previous reports, ranging from 4-11 days. We repeated the angiogram every week to check for the changes over time. In previous literature, the fastest detection of the time point of entire recanalization was 14 days from the first discovery of disappearance. Although it is difficult to draw a definite conclusion, it seems appropriate to conduct the first follow-up angiogram within 14 days after detection of spontaneous disappearance.

CONCLUSION

The exact timing of the recanalization following the spontaneous thrombosis of the ruptured aneurysm is difficult to predict. Repeated angiogram is crucial for the surveillance of recanalization.

NOTES

Conflict of interest
No potential conflict of interest relevant to this article was reported.

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