Rebleeding From a Vertebral Artery Dissecting Aneurysm After Endovascular Internal Trapping: Adverse Effect of Intrathecal Urokinase Injection or Incomplete Occlusion?—Case Report—

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Abstract

A 67-year-old woman suffered rebleeding from a ruptured vertebral artery dissecting aneurysm after endovascular internal trapping. The dissecting aneurysm was initially successfully occluded with the affected vertebral artery using detachable coils. However, rebleeding from the aneurysm occurred on the next day. The rebleeding may have resulted from the thrombolytic effect of urokinase, which was injected intrathecally 3 hours before rebleeding occurred, or the relatively loose coil packing of the aneurysm. This case indicates the potential risk of intrathecal use of thrombolytic agents and the importance of complete tight coil packing of the whole dissected site in the treatment of ruptured vertebral artery dissecting aneurysms.

Key words: coil, endovascular treatment, internal trapping, rebleeding, thrombolytic agent
Introduction

Intracranial vertebral artery dissecting aneurysms (VADAs) have recently been recognized as an important cause of subarachnoid hemorrhage (SAH). The rebleeding rate in the acute stage following SAH is very high, so ultra-early intervention is strongly recommended in the treatment of ruptured VADAs. Endovascular neurosurgeons initially treated ruptured VADAs by occluding the parent artery at or just proximal to the dissecting site using balloons or fibered platinum coils. Since the introduction of the Guglielmi detachable coil (GDC), endovascular treatment has become safer and more effective because the detachable coil is soft and more easily controlled. The emphasis of endovascular treatment then shifted from proximal occlusion to internal trapping, in which the dissected site is completely occluded with detachable coils. This procedure can completely exclude the aneurysm from the circulation and theoretically carries no risk of rebleeding.

We report a case of rebleeding after endovascular internal trapping of the ruptured VADA and discuss the mechanism of rebleeding.

Case Report

A 67-year-old woman was admitted to our hospital 4 hours after sudden onset of headache. Computed tomography (CT) revealed SAH. Emergent digital subtraction angiography showed a right VADA (Fig. 1 left).

A 6-Fr guiding catheter was introduced into the right VA with the patient under general anesthesia immediately after diagnostic angiography. An Excel-14 microcatheter (Boston Scientific, Fremont, Calif., U.S.A.) was navigated coaxially into the VADA. Nine GDCs (Boston Scientific) were placed in the aneurysm, which resulted in successful occlusion of the aneurysm with the affected VA. Postoperative angiography revealed complete occlusion of the right VA proximal to the aneurysm and good collateral flow to the posterior circulation through the left VA without retrograde flow into the aneurysm (Fig. 1 right). Lumbar drainage was then placed, and she showed good recovery from general anesthesia. Urokinase 6000 U was injected though the lumbar drainage catheter to wash the subarachnoid clot during the following morning (15 hours after initial onset), because CT showed massive SAH (Fisher group III). She suddenly complained of severe headache 3 hours after the injection of urokinase, and became comatose soon after. Emergent CT demonstrated repeated SAH.

With the patient under general anesthesia, emergent angiography showed complete occlusion of the right VA, and left vertebral angiography demonstrated extravasation of contrast medium at a site distal to the aneurysm (Fig. 2). A 6-Fr guiding catheter was introduced into the left VA. A ProGreat microcatheter (Terumo, Odawara, Kanagawa) was navigated coaxially through the left VA into the distal
part of the aneurysm in the retrograde direction. Four GDCs were tightly placed in the aneurysm and the distal part of the right VA, which resulted in complete exclusion of the aneurysm from the circulation (Fig. 3). The patient’s neurological condition improved gradually after the second procedure. She was transferred to another hospital to continue rehabilitation 2 months after onset. Severe tetraparesis persisted at the time of discharge.

**Discussion**

The typical angiographical finding of VADAs is the “pearl-and-string” sign. Entry-exit cerebral dissecting aneurysms are historically well known as a common type of cerebral dissection, but the majority of ruptured VADAs have entry-only dissection, of which the pseudolumen is the cul-de-sac and rebleeding can easily occur during the acute stage. Complete coil packing of the whole dilated part including the affected VA (internal trapping) is therefore recommended as an endovascular procedure to prevent rebleeding in the acute stage following SAH. We have treated 60 patients with ruptured VADAs using endovascular techniques between 2000 and 2008. Forty-nine of the 60 patients were treated within 24 hours of onset. All except two patients were treated by internal trapping using detachable coils. The present case was the only one complicated with rebleeding from the ruptured VADA after endovascular internal trapping. We suspect that relatively loose packing of the distal part of the “pearl” aneurysm, from which extravasation of the contrast medium was seen at second angiography, may have caused the rebleeding. The packing density in the aneurysm in the present case was not particularly dense but was judged to be adequate to prevent rebleeding. We treated several cases with looser coil packing, but no rebleeding occurred in any of these other cases. However, the present case suggests that coil packing of the aneurysm must be as dense as possible to prevent rebleeding. Recanalization of the VADA may occur after internal trapping, which emphasizes the importance of dense packing of the coils during endovascular treatment for VADA.

Intrathecal injection of urokinase was certainly a more important issue. Rebleeding occurred 3 hours after urokinase injection. This was the first and only case in which we injected urokinase intrathecally in our series of 60 cases of ruptured VADAs. We strongly suspect that the thrombolytic effect of the urokinase might have been responsible for the rebleeding, because the arterial wall in VADA is histologically very thin. Interestingly, in a similar case, the patient died of rebleeding one week after proximal occlusion of a VADA, and tissue plasminogen activator was used to clear the patient’s ventriculostomy catheter, although whether this injection was related to rebleeding remained unclear. These two cases demonstrate the potential risk of intrathecal use of thrombolytic agents during the treatment of ruptured VADA even if endovascular occlusion has been achieved. We have not used thrombolytic agents to clear cerebrospinal fluid in subsequent cases. We would like to emphasize that intrathecal injection of thrombolytic agents should not be given if only incomplete coil packing was achieved in the endovascular treatment for ruptured VADA.

**References**


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