

Glue Embolization of the Giant Aneurysm by Reducing Thrombosis-Induced Volume Expansion Effect

혈전형성에 의한 부피팽창을 감소시키기 위한 거대동맥류의 아교색전술

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A giant aneurysm due to a large intra-aneurysmal volume can be complicated by a delayed massive volume expansion caused by thrombus formation. To prevent such a severe mass effect, we obliterated the aneurysmal lumen by gluing and prevented further development of thrombosis. A 52-year-old female with a giant aneurysm at the cavernous segment of the internal carotid artery presented with tinnitus and intermittent diplopia. After confirming with a negative occlusion test, the right internal carotid artery was trapped by coiling and with further obliteration of the aneurysmal lumen by gluing. She developed a mild diplopia after the procedure and recovered without any deficit. The magnetic resonance angiography showed a stable occlusion of the aneurysm and good collateral filling of the cerebral vessel 15 months later.

Index terms

Giant Aneurysm
Cerebral Aneurysm
Glue Embolization

INTRODUCTION

Large or giant aneurysms in the cavernous segment of the internal carotid artery (ICA) frequently present with neurological symptoms caused by the mass effect on the cranial nerves. Thus, the treatment goal for a symptomatic giant ICA aneurysm in the cavernous segment is resolution of the mass effect that leads to cranial nerve dysfunctions (1). Several endovascular treatments for giant aneurysms have already been introduced, such as detachable coiling and flow diverter stent insertion. However, complete packing of the aneurysmal sac itself is difficult, and the recurrence rate is high (2). These endovascular techniques can also cause transient swelling of the aneurysmal sac after induced thrombosis as well as delayed complications, including bleeding. In this case report, we described our first use of surgical n-butyl 2-cyanoacrylate (NBCA) and coils for treating a symptomatic, giant saccular aneurysm located in the cavernous segment of the ICA.

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CASE REPORT

A 52-year-old female presented with tinnitus and intermittent diplopia. A cerebral angiography showed a giant aneurysm ($31.4 \times 15.7 \times 17.5$ mm) arising in the cavernous segment (C4, ascending segment) and protruding medially (Fig. 1A). An initial magnetic resonance imaging (MRI) showed a partially thrombosed, giant aneurysm at the level of the right cavernous sinus and protruding into the sphenoid sinus. The patient underwent a balloon occlusion test of the right ICA for 20 minutes, and there was no focal neurologic sign or lateralizing sign seen during the occlusion test. A single photon emission CT using ^{99m}Tc hexamethylpropyleneamineoxime injected during the balloon inflation demonstrated no cerebral perfusion alteration.

Following the balloon occlusion test, the right ICA was trapped by coiling at two points, i.e., just proximal and distal to the aneurysm. After isolating the aneurysm with a complete ICA occlusion, a 25% NBCA-lipiodol mixture was injected into the aneu-

rismal sac in order to prevent volume expansion by thrombosis (Fig. 1B, C), and additional coil packing at the petrous and cervical ICA was done. Finally, stump occlusion using a 6 mm Ampatzter vascular plug was done in the cervical ICA.

Immediately after the procedure, the patient developed partial sixth nerve palsy, presenting with mild diplopia, which was completely resolved with an anti-inflammatory medication. A follow-up MRI done 15 months later showed the decreased size of the thrombosed aneurysm in the source magnetic resonance angiography (MRA) image compared to the initial MRA image (Fig. 1D, E). There was good filling of the ipsilateral cerebral vessels with an obliteration of the right ICA and the aneurysm (Fig. 1F). The patient did not express any complaints and did not re-

veal any neurological deficit during the follow-up period.

DISCUSSION

Many giant aneurysms present with symptoms caused by their mass effect. The exact clinical manifestation of the mass effect depends on the location of the aneurysm and the involved adjacent neural structures. Giant aneurysms arising from the cavernous and proximal intracranial carotid arteries present with dysfunction of ocular movement as well as vision deterioration (3, 4). During the past 30 years, several endovascular treatments for giant cavernous ICA aneurysm have been introduced, such as detachable coiling and flow diverter stent insertion. Detach-

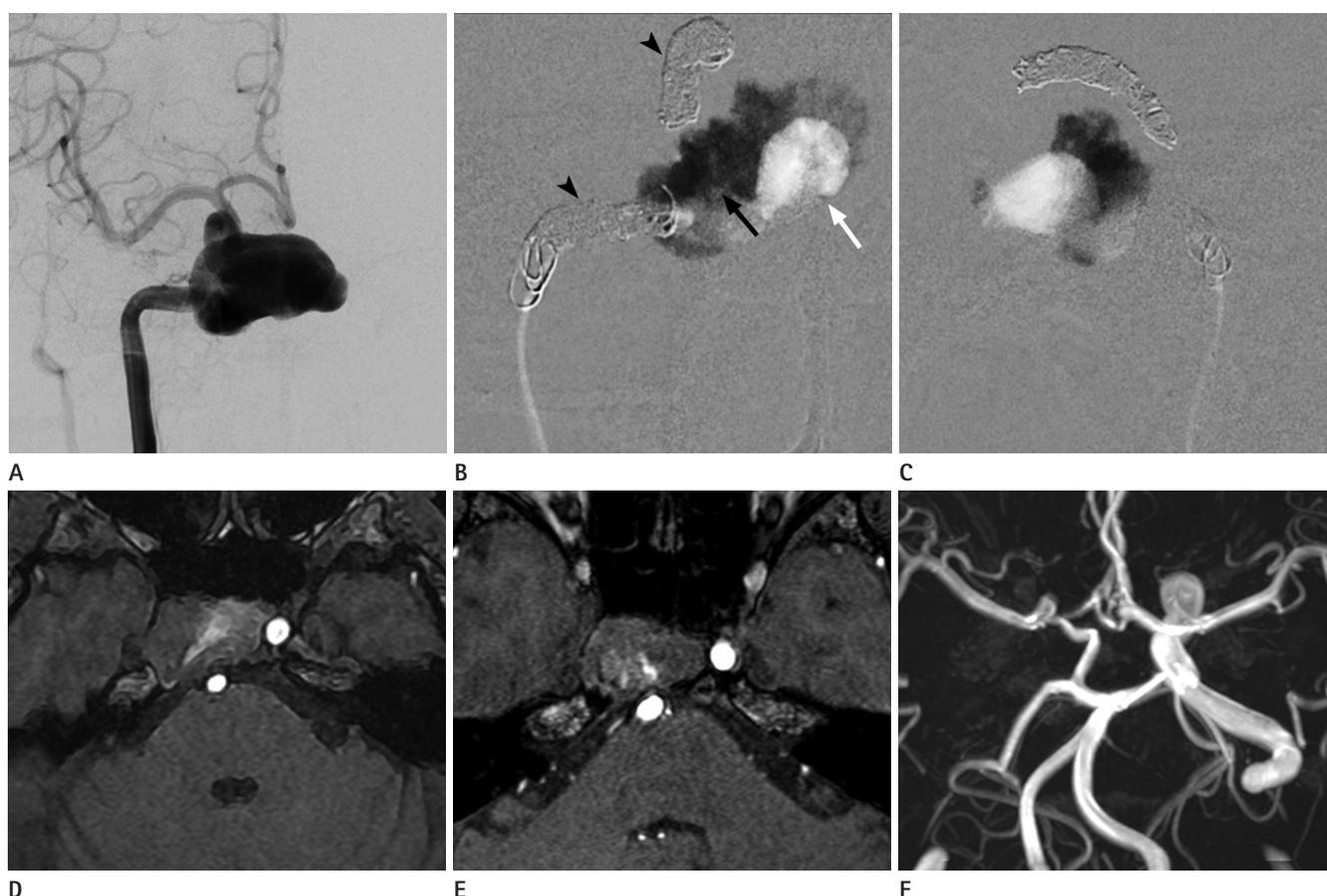


Fig. 1. A 52-year-old female presented with tinnitus and intermittent diplopia.

A. The right internal carotid arteriogram showed a giant aneurysm at the end of the petrous segment protruding sphenoid sinus medially. **B, C.** After confirming negative occlusion test, the right internal carotid artery (ICA) was trapped by coiling (arrowheads) with further obliteration of the aneurysmal lumen by gluing to prevent volume expansion of thrombus which may lead to massive nasal or even intracranial bleeding. Note migration or spreading of the glue from the initial injection site (white part indicated by white arrow) to the final position (black part indicated by black arrow).

D, E. Note decreased size of thrombosed aneurysm in the source image of magnetic resonance angiography (MRA) (**E**) 15 months later compared to the initial MRA (**D**).

F. Good filling of ipsilateral cerebral vessels with obliteration of the right ICA and aneurysm.

able coils can be used for packing of the aneurismal sac itself or of the parent artery (5). Flow diverter stents are used to restrict and redirect blood flow along the vessel axis for reconstructing the parent vessel lumen across the aneurysm neck, thus promoting aneurysm thrombosis (6).

These endovascular techniques can, however, lead to unfavorable clinical outcomes, including ischemia and compression syndromes caused by transient aneurysm swelling after an induced sac thrombosis and subsequent bleeding of a previously un-ruptured aneurysm (7). In addition, the incidence of recanalization and incomplete embolization of the giant aneurysm lumen is high due to the difficulty of compact coil packing for the large volume (2).

In order to obliterate dead space in the aneurysmal sac, which is subject to being thrombosed, we used a 25% NBCA-lipiodol mixture after coil packing of the parent arterial lumen. We expected that the penetration of glue in the aneurysm could prevent it from migrating distally through the coil mesh in the completely occluded distal ICA lumen. *In vitro* studies of the 28% glue concentration in a previous study, sticking of the micro-catheter to the glue cast did not occur (8). In addition, there was no resistance during the retrieval of the micro-catheter.

The aneurysm size may decrease after NBCA embolization, as seen on the follow-up MRI. It seemed to result from three postulated mechanisms as follows (8, 9): 1) NBCA in the aneurysm is absorbed by the blood; 2) NBCA in the aneurysm is absorbed through the aneurysmal wall after an inflammatory process caused by the NBCA; or 3) there is extra-luminal migration of the NBCA through the aneurysmal wall. On this basis, the transient diplopia that occurred in our patient can be explained by an inflammatory process in the aneurysmal wall, with the use of 25% concentration NBCA. A future study to find out the exact mechanism of this phenomenon is required. We believe that the decrease in the aneurysm size after a NBCA embolization can result in a mass-relieving effect, which can also be achieved by a parent artery occlusion (10).

Our report has several limitations for further considerations. First, we could not completely obliterate the mass effect using NBCA, even though the mass effect was not serious. Therefore, it would be necessary to both predict and manage any further mass effect by using steroids, for example. Secondly, the injected low concentration of NBCA slowly had contact with the blood

in the aneurysm and slowly spread the coagulation effect during the real-time fluoroscopy, thus suggesting a potential effect of volume expansion. Therefore, a slow injection of NBCA is required with careful real-time fluoroscopic observation of the direction and extent of the NBCA.

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혈전형성에 의한 부피팽창을 감소시키기 위한 거대동맥류의 아교색전술

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거대동맥류(giant aneurysm)의 혈관내치료 후 혈전형성은 덩어리효과(mass effect)로 인한 심각한 합병증을 가져올 수 있다. 혈전형성에 동반되는 합병증을 감소시키기 위해 거대 동맥류의 내강을 수술용아교(n-butyl 2-cyanoacrylate)로 폐쇄하여 혈전발생공간을 줄이는 치료 보고는 거의 없었다. 이명과 복시로 내원한 52세 여자 환자에서 내경동맥의 해면정맥동 분절(cavernous segment)에 거대동맥류를 발견하였다. 폐쇄실험(occlusion test)의 음성결과를 확인하고 동맥류 전후의 내경동맥을 코일(coil)로 폐쇄하여 동맥류를 고립시킨 후 동맥류의 내강을 수술용아교로 폐쇄하였다. 시술 후 환자는 일시적 복시를 호소하였으나 15개월 후 시행한 MR 혈관 조영상 동맥류 내강의 안정적 폐쇄 및 잘 발달한 측순환(collateral flow)을 확인함으로써 거대동맥류의 내강을 수술용아교로 폐쇄하여 혈전형성에 의한 덩어리효과를 최소화할 수 있었다.

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