Management of a Direct Carotid Cavernous Fistula Caused by Rupture of a Cavernous Aneurysm Previously Embolized with Coils

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Summary

The development of a high-flow carotid-cavernous fistula from the rupture of a large cavernous aneurysm successfully embolized by coils is rare. A 50-year-old male patient developed a high-flow carotid-cavernous fistula 48 hours after successful coiling of a large left cavernous aneurysm, presumably due to rupture of a focal dissection at or close to the neck of the aneurysm. He initially responded to daily self-compression of the left common carotid artery, but the fistula recurred. After failing to approach the fistula site via transvenous route, balloon trapping of the internal carotid artery was planned. Prior to its placement for functional occlusion test, the detachable balloon slipped into the fistula site and occluded it. It was thereafter detached in this position. The sequence of events, a large cavernous aneurysm spontaneous ruptured after coiling, suggested dissecting process or disease. We address in the report the complexity of the endovascular management of this rare association.

Introduction

Cavernous aneurysms constitute approximately 5% of all intracranial aneurysms. 16% of them are giant aneurysms¹ and there is a female preponderance. Most of the cavernous

aneurysms detected during angiography are incidental lesions without causing any symptoms, and they usually warrant no further treatment². Symptoms are most commonly due to the mass effect of the aneurysms, causing headache and cranial nerve palsy³. Rupture of a cavernous aneurysm will result in the development of a direct carotid-cavernous fistula (CCF), but this is an uncommon event⁴. Transarterial occlusion of the direct CCF by detachable balloon is usually the treatment of choice because the efficacy and safety of this technique have been well established¹⁷. However, in cases which transarterial approach is infeasible or carrying a higher risk, the transvenous route should be attempted¹⁰. We report a rare case in which a male patient suffered from rupturing of a cavernous aneurysm 48 hours after embolization with coils, resulting in a direct high-flow CCF.

Case Report

A 50-year-old man was referred for further management of progressive left sixth nerve palsy caused by a large left cavernous aneurysm. He had history of hypertension, but there is no history of recent trauma, no family history of collagen disorder. The aneurysm was measured 16mm x 14mm in size, located at the junction of C4 and C5 segments, and had a neck-to-fundus ratio which was favorable for embolization

Figure 1 (A) Frontal and (B) lateral views of the left internal carotid angiogram showing the large aneurysm at the cavernous segment of the left internal carotid artery before embolization with coils. (C) Frontal, (D) lateral and (E) 3D reconstruction views of the same cavernous aneurysm immediately post-embolization, demonstrating near-complete obliteration of the aneurysmal lumen.

with coils. We successfully achieved near-complete embolization of this cavernous aneurysm with Guglielmi detachable coils 18 (GDC 18, Target Therapeutics, Fremont, CA) under general anesthesia (figure 1). The procedure was uneventful. Patient remained well immediately after the embolization procedure. However, 48 hours later, he developed sudden onset of pulsatile tinnitus and on clinical examination, he had left-sided proptosis, chemosis, and new onset of left third nerve palsy on top of his pre-existing sixth nerve palsy. A follow-up cerebral angiogram demonstrated a direct high-flow left CCF associated with a newly developed aneurysmal pouch just anterior to the coils, which could represent a dissecting aneurysm (figure 2). There is no initial flap detectable.

The coils inside the original cavernous aneurysm showed no significant change in architecture. The high-flow CCF drained into the ipsilateral superior ophthalmic vein, the contralateral cavernous sinus, and inferiorly to the pterygoid venous plexuses and bilateral inferior petrosal sinuses. There was mild cortical venous reflux into the posterior fossa identified. We advocated conservative treatment strategy by daily self-compression of the left common carotid artery at that juncture, in view of a potential for healing for this post-procedural complication. In the subsequent weeks, he showed significant improvement of his third and sixth nerve palsy and gradual amelioration of his left-sided proptosis and chemosis, as well as pulsatile tinnitus. The patient was advised to continue to perform daily self-compression of his left common carotid artery and he was discharged back home with an outpatient follow-up appointment arranged.

The patient remained clinically stable until ten weeks after the first embolization procedure when he developed recurrence of left-sided proptosis, chemosis, periorbital edema, left third nerve palsy and a new onset of left fourth nerve palsy. The visual acuity of his left eye was decreased associated with persistent retro-orbital pain. A repeat cerebral angiogram demonstrated a direct high-flow left CCF that drained into the ipsilateral superior ophthalmic vein, the contralateral cavernous sinus, the pterygoid venous plexuses, and bilateral inferior petrosal sinuses. There was no significant cortical venous reflux into the posterior fossa observed this time. The coils did not show significant

Figure 2 (A) Frontal and (B) lateral views of the left internal carotid angiogram showing the high-flow CCF developed from the rupture of the coil-embolized cavernous aneurysm.

compaction or change in conformation; however, an aneurysmal pouch was still noted anteroinferior to the coil mesh. In view of a possibility of a dissecting process of the left internal carotid artery, we initially attempted transvenous route to exclude the CCF, with a hope to avoid sacrificing the internal carotid artery. We failed to advance the catheter into the venous compartment of the CCF either via the contralateral cavernous sinus, the ipsilateral inferior petrosal sinus, or the ipsilateral facial vein. After these attempts of catheterization, the procedure was discontinued and a control cerebral angiogram demonstrated that the CCF

Figure 3 (A) Frontal and (B) lateral views of the left internal carotid angiogram performed ten weeks after the first embolization procedure, demonstrating the recurrence of the CCF after temporary healing in response to daily self-compression of the left common carotid artery.

was no longer opacified. Despite these aspects, the patient was scheduled for a balloon occlusion test the next day prior to sacrificing the left internal carotid artery by balloon trapping. However, a repeat cerebral angiogram showed complete re-opening of the CCF 24 hours later (figure 3). A #16 Gold Valve detachable balloon was used for the test. On inflating the balloon at the site just proximal to the CCF, it suddenly and unexpectedly slipped into the venous side of the fistula. The balloon was further inflated until complete exclusion of the fistula

while preserving full patency of the carotid artery. We detached the balloon at this position and a control cerebral angiogram demonstrated total occlusion of the CCF and no contrast opacification of the previous cavernous aneurysm (figure 4,5).

The left internal carotid artery was completely patent. Subsequent clinical follow-up revealed marked improvement of the visual acuity, retro-orbital pain, left-sided proptosis, chemosis and periorbital oedema. There was no transcranial or transorbital bruit.

Figure 4 (A) Frontal and (B) lateral views of the left internal carotid angiogram immediately after successful occlusion of the CCF by the detachable balloon. Note that the possible dissecting aneurysm is no longer opacified, and the parent artery is completely patent.

Figure 5 (A) Frontal and (B) lateral radiographs of the skull showing the position of the detachable balloon within the left cavernous sinus, and its relationship with the coil mesh inside the original cavernous aneurysm.

Discussion

The surgical treatment of cavernous aneurysm is difficult because of the surrounding cavernous sinus and its close proximity to the four cranial nerves. The therapeutic options in the past included surgical ligation of the internal carotid artery and direct microsurgical exposure of the cavernous aneurysms^{5,6}. In recent years, endovascular treatment strategies are replacing the surgical treatments because they are less invasive, less time consuming and having fewer complications⁷. Balloon occlusion of the parent vessel and endosaccular coiling are the two frequently employed methods. For patient with cavernous aneurysm that has favorable neck-to-fundus ratio and has strong clinical indications to preserve the patency of the parent internal carotid artery, endosaccular coiling becomes the treatment of choice. A recent systemic review on the efficacy and safety of the endovascular treatment of cavernous aneurysms demonstrated that endosaccular coiling could achieve >90% occlusion of the aneurysms in 80% of the patients, and none of them had a reported permanent complication⁷. Our patient is relatively young (50 years old) and had a history of hypertension. We did not want to sacrifice his internal carotid artery by balloon occlusion. Therefore, we advocated endosaccular coiling for his narrow-necked cavernous aneurysm at the first instance.

Rupture of a cavernous aneurysm after successful embolization with GDC coils is a rare complication. In the aforementioned systemic review on the endovascular treatment of the cavernous aneurysms, which involved 35 studies published between January 1974 and May 1999, there was no such complication reported⁷. We believed that the rupture could be originated from a dissecting injury either caused or triggered by the previous procedure, as suggested by the development of a new aneurysmal pouch adjacent to the neck of the pre-existing cavernous aneurysm. This resulted in the development of a CCF, which could have been traumatic in nature. As there should be a potential for healing for the traumatic type of CCF, we initially attempted conservative treatment by instructing the patient to perform daily self-compression of the ipsilateral common carotid artery. This maneuver slowed down the blood-flow across the CCF and thus promoting thrombosis and spontaneous closure of the fistula. Successful treatment of a traumatic, direct CCF by this method had been reported⁸. The patient did response to conservative treatment initially, with gradual improvement of his proptosis, chemosis and cranial nerve palsy. Unfortunately, his CCF re-opened subsequently after ten weeks.

The re-establishment of his left-sided direct high-flow CCF on top of a post-coiling cavernous aneurysm associated with a suspected dissecting aneurysm at or close to its aneuris-

mal neck created a challenging clinical condition. We considered two viable treatment options: (i) by embolization of the CCF with coils via a transvenous route, thus avoid further manipulation of the presumed traumatized artery as well as preserving the arterial patency, (ii) by endovascular trapping of the left internal carotid artery.

Embolization of a direct type of CCF with coils via a transvenous route had been described as an alternative for cases in which the conventional technique failed or had increased risk^{9,10,16}. The often-utilized transvenous accesses to the cavernous sinus are the inferior petrosal sinus, the superior ophthalmic vein, the pterygoid plexus and the intercavernous sinus via the contralateral cavernous sinus¹¹⁻¹³. We decided to utilize the transvenous route in order to preserve the parent vessel as well as to avoid further possible trauma to the artery, but this was proved to be difficult. The relatively low success rate of approaching the fistula site via the transvenous route had also been emphasized by others^{14,15}.

The successful occlusion of the CCF by a #16

Gold valve detachable balloon that slipped into the fistula site during balloon occlusion test demonstrated that closure of the fistula by a detachable balloon could be a viable option in the management of such a rare condition, in case a safer transvenous route failed. Post-procedural close monitoring is important to detect complications such as prolapse of the balloon that could lead to narrowing or even occlusion of the internal carotid artery, as well as premature balloon deflation leading to recurrence of CCF¹⁵.

Conclusions

Rupture of a successfully embolized cavernous aneurysm resulting in a high-flow CCF is a rare but challenging clinical condition. It could be related to dissection either caused or triggered by the endovascular treatment.

Transvenous approach to treat such CCF has a theoretic advantage of avoiding further arterial injury, but transarterial balloon occlusion of the fistula could be considered as an alternative in case it failed.

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