Spontaneous Thrombosis of a High-flow Carotid-Cavernous Fistula after Failed Transarterial Balloon Occlusion

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Summary

A 62-year-old man with a traumatic high-flow right carotid-cavernous fistula was treated by transarterial balloon occlusion technique. However, because of the relatively small size of the fistula, the balloon could not enter into the cavernous sinus via the fistula. During the procedure, the shunt flow decreased significantly, and we stopped the procedure. Follow-up angiography performed 14 days after the procedure showed complete occlusion of the fistula with a small residual pseudoaneurysm. One year later, the pseudoaneurysm had decreased in size. Repeated transient decrease and stagnancy of blood flow at the fistula during the balloon procedure may have played an important role in the thrombosis in this patient.

Introduction

The direct type of high-flow carotid-cavernous fistula (CCF) is traditionaly treated by transarterial detachable balloon occlusion technique [1,2]. Recently, Guglielmi detachable coils (GDCs) were used for the treatment of direct CCF [3,4]. The transvenous approach can also be used when the transarterial approach is unsuccessful [5,6]. Direct CCF rarely closes spontaneously [7,8], and low-flow direct CCF is sometimes treatable by self-compression of the common carotid artery [9]. We experienced a case of high-flow CCF that thrombosed spontaneously after failure of transarterial balloon occlusion technique.

Case Report

A 62-year-old man suffered a traffic accident resulting in multiple facial bone fractures. Five days after the accident, right abducens nerve palsy developed that caused double vision. Right chemosis, exophthalmos, and orbital bruit also developed gradually. Magnetic resonance images obtained 43 days after the head trauma showed right-side dominant bilateral chronic subdural hematomas with dilated right cavernous sinus and superior ophthalmic vein, indicative of the presence of a CCF. Subsequently, selective cerebral angiography was performed, and right high-flow direct CCF was confirmed (figure 1). Simultaneously performed balloon occlusion Matas test was positive (i.e., intolerable). Six days later, transarterial occlusion of the fistula with a detachable balloon was attempted under systemic heparinization through a 9F guiding catheter.

The slightly inflated balloon (#9 Goldvalve, Nycomed, Paris, France) seated in the orifice many times but never entered the cavernous sinus via the fistula because the fistula was relatively small. During the procedure, blood flow

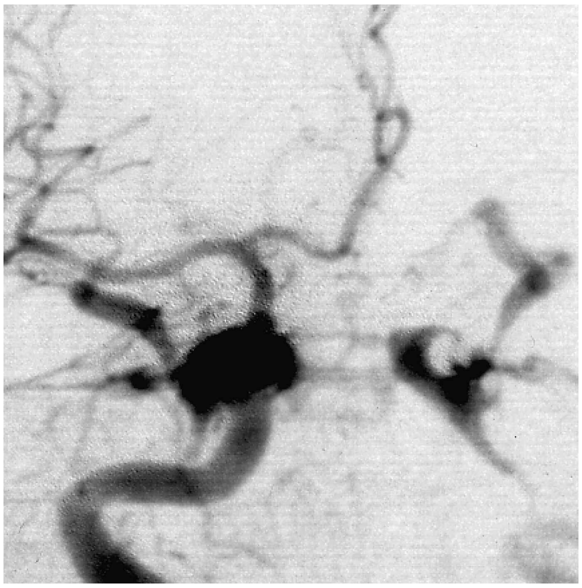

Figure 1 Anteroposterior projection of selective right internal carotid arteriography shows a high-flow direct carotid-cavernous fistula. The anterior cerebral arteries are compressed to the left by the right subdural haematoma.

in the internal carotid artery (ICA) decreased because of the large size of the guiding catheter and catheterization-related vasospasm, and we found that the shunt flow decreased significantly (figure 2). Therefore, we stopped the procedure. Over the following days, the orbital bruit disappeared, and right chemosis and exophthalmos improved. Follow-up angiography performed 14 days after the procedure showed occlusion of the fistula with a small residual pseudoaneurysm (figure 3). The size of the pseudoaneurysm had decreased one year later (figure 4). There is no evidence of recurrence of the CCF at three years after the procedure.

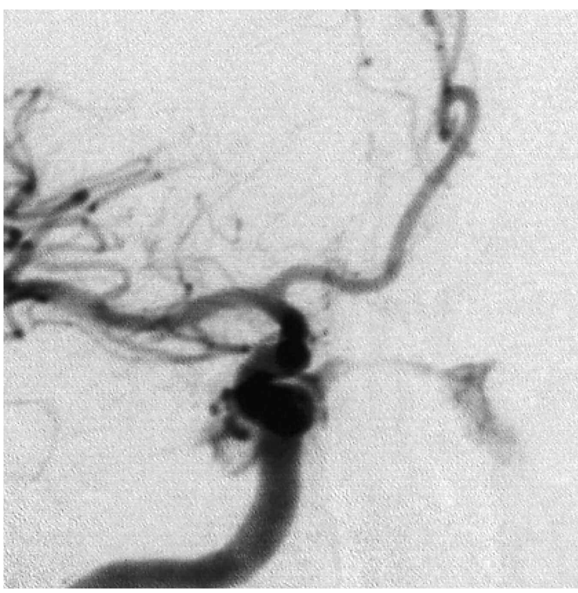

Figure 2 Anteroposterior projection of selective right internal carotid arteriography during transarterial balloon occlusion shows significant decrease of the shunt flow.

Discussion

According to Barrow et Al [10], CCF is classified into four types: type A involves a direct shunt between the intracavernous ICA and the cavernous sinus (CS); types B, C, and D involve indirect transdural shunts between the CS and dural branches of the ICA, external carotid artery or both. The majority of type A (direct) CCFs occur as a rare complication of head injury with basal skull fracture, and a minority occur because of rupture of an intracavernous ICA aneurysm, resulting in a high-flow shunt [11]. Another risk factor for direct CCF is connective tissue disorders [12]. Spontaneous dissection is also a rare cause of direct CCF [13]. In the present patient, the CCF was caused by trauma. Rupture of a preexisting small aneurysm, triggered by the rupture caused by the trauma, may also have occurred. However, the small aneurysm decreased in size one year later, indicating that it was not a true aneurysm but a pseudoaneurysm [14].

Endovascular closure is the treatment of choice in direct CCF. Endovascular balloon occlusion was first described by Serbinenko [1] and popularized by Debrun et Al [2]. Usually, a transarterial approach is attempted. If it fails, a transvenous approach is the alternative [5]. Recently, GDCs have been used for the treatment of direct CCF via both the transarterial and transvenous approaches [4]. Even though a fistula is small in caliber, it can be treated with GDCs because it is technically easier to guide a microcatheter and microguidewire combination through a small fistula than it is to guide a balloon [3]. Thus, in hindsight, we should have used GDCs rather than a balloon to treat the direct CCF in our patient.

Direct CCFs sometimes thrombose spontaneously [7,8]. Mechanisms thought to be responsible for the spontaneous thrombosis of direct CCF are slow flow due to ICA dissection leading to venostasis and damage to the vascular

Figure 3 Anteroposterior projection of right common carotid arteriography performed 14 days after the balloon occlusion procedure shows occlusion of the fistula and a small residual pseudoaneurysm at the medial wall of the cavernous internal carotid artery.

Figure 4 Anteroposterior projection of right common carotid arteriography performed approximately one year after the procedure shows the residual pseudoaneurysm has decreased in size.

lining of the CS caused by venous hypertension 8. Nishijima et Al [7] reported a case of spontaneously healed direct CCF immediately after orbital venography and speculated that a temporary decrease in the pressure gradient between the cavernous sinus and the ICA caused formation of a thrombus. Spinnato et Al [9] reported a case of a direct slow-flow CCF successfully treated by self-compression of the common carotid artery. Compression induces simultaneous arterial hypotension and venous hypertension, which result in a transient decrease of the pressure gradient across the shunt, thus promoting thrombosis. The present patient had a high-flow shunt, but the blood flow in the ICA decreased during the intraarterial procedure because we used a 9F guiding catheter and because of vasospasm related to the catheterization.

Moreover, the slightly inflated balloon seated in the orifice of the fistula many times, resulting in intermittent occlusion of the fistula. These transient reductions in blood flow and the resulting stagnancy may have played an important role in the thrombosis of the CCF in the present patient. Thus, had it been performed, self-compression of the common carotid artery, which produces a similar reduction in blood flow, might also have been useful for the treatment of the direct CCF in the present patient. Another explanation of spontaneous thrombosis in this patient may be that the injection of iodinated contrast media exaggerated leukocytic accumulation, promoted red blood cell aggregation, and directly affected the vascular endothelium [8].

Conclusions

Repeat transient decrease and stagnancy of blood flow at the fistula may have played an important role in the thrombosis of the CCF in the present patient. Thus, we suggest that temporary occlusion of the orifice of the CCF with a balloon may produce permanent thrombosis of the fistula. For a similar reason, self-compression of the common carotid artery may possibly be useful for the treatment of the direct CCF with a relatively small fistula. If endovascular therapy is considered, GDCs appear to be a better choice than are balloons for the treatment of a direct CCF.

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EDITORIAL COMMENT

The authors report secondary thrombosis of carotico-cavernous fistula which does not seem to be of very high flow if one considers the satisfactory opacification in the intracranial arteries. The flow was certainly not high enough to allow a large balloon to penetrate inside the fistula and as suggested by the authors one can think that several inflations at the level of the small communication led to some further traumatic changes and secondary clotting. However we must insist on the fact that manual neck compression should never be recommended in such a situation; it would certainly produce a steal phenomenon from the intracranial circulation down to the ipsilateral fistula. Deficit would certainly occur rapidly and could very well be severe if the patient had compressed his neck with the ipsilateral hand. Similarly balloon testing should not be performed below the fistula but at the level of the fistula or distal to it in order to assess properly the capacity of the circle of Willis.

In general, numbers 9, 12, 16 Gold-valve balloon (GVB) could be used in large fistulae and numbers 7, 17 and 19 that inflate from tip to base should be used in priority for small hole or inferior hole fistulae. Guiding catheters with GVB are routinely 7 and 8F; 9 French, particularly in the Asian population are most often not needed and could very well be traumatic for the neck vessel.

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