CASE REPORT

Embolization of a ruptured lenticulostriate artery aneurysm

Embolisation d’un anévrisme rompu de l’artère lenticulostriciée

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KEYWORDS
Lenticulostriate artery; Aneurysm; Embolization

Summary Aneurysms arising from the lenticulostriate artery (LSA) are rare. So far, only 23 cases have been reported in the literature (Ahn et al. 2007[1], Gandhi et al. 2008[2], Harreld et al. 2010[3]). Early detection and treatment of these aneurysms is difficult because of their small size, deep location and complex surrounding vasculature. The majority of reported cases were treated surgically, and only two were treated with endovascular embolization (Harreld et al. 2010[3], Larrazabal et al. 2001[4]). We present here a case of an LSA aneurysm that was successfully embolized with n-butyl cyanoacrylate (n-BCA) with no recurrence after 1 year of follow-up.

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Introduction

Aneurysms arising from the lenticulostriate artery (LSA) are rare. So far, only 23 cases have been reported in the literature[1—3]. Early detection and treatment of these aneurysms is difficult because of their small size, deep location and complex surrounding vasculature. The majority of reported cases were treated surgically, and only two were treated with endovascular embolization[3,4]. We present here a case of an LSA aneurysm that was successfully embolized with n-butyl cyanoacrylate (n-BCA) with no recurrence after 1 year of follow-up.

Case report

A 51-year-old woman experienced sudden-onset headache followed by disorganized speech and general weakness. The patient had no history of hypertension, intracranial disease, recent infectious disease, head injury or drug abuse. On admission, her initial Glasgow Coma Scale (GCS) score
was 15. Computed tomography (CT) revealed an intracerebral hemorrhage in the left medial temporal region, with rupture into the temporal horn of the lateral ventricle and moderate hydrocephalus (Fig. 1). CT angiography demonstrated stenosis of the left middle cerebral artery (MCA), suggesting vasospasm, and an aneurysm in the medial temporal region that was anatomically correlated with the intracerebral hemorrhage (Fig. 2). Conventional angiography demonstrated diffuse vasospasm with an aneurysm 4 mm in diameter arising from the distal portion of the left medial LSA (Fig. 3).

The patient was referred for endovascular treatment 3 days after symptom onset. Transarterial embolization was accomplished with a standard Seldinger puncture through the right femoral artery. A 6-F Envoy guiding catheter (Cordis Corporation, Miami Lakes, FL, USA) was advanced over a 0.035-inch guidewire (Terumo Corporation, Tokyo, Japan) into the left internal carotid artery (ICA), through which an Excelsior SL-10 microcatheter (Boston Scientific, Natick, MA, USA) was advanced over a Transcend-14 microguidewire (Boston Scientific) such that the catheter tip lay within the LSA aneurysm. Superselective contrast injection was then performed to confirm the position of the catheter and the aneurysm (Fig. 4). Next, a 33.3% n-BCA mixture, prepared with 0.5 mL of n-BCA (Ingenor, Gennevilliers, France) and 1.0 mL of ethiodized oil (Lipiodol Ultra Fluid; Guerbet, Aulnay-sous-Bois, France), was infused to fill the aneurysm. Complete aneurysm obliteration was identified on angiography performed immediately after embolization (Fig. 5).
Figure 5  Left oblique internal carotid angiography immediately after embolization demonstrates complete obliteration of the aneurysm with preservation of the parent artery. Note that vasospasm is still present.

Figure 6  Left oblique internal carotid angiography 12 months after embolization demonstrates complete obliteration of the aneurysm with no recurrence.

The patient had an uneventful postoperative recovery and was discharged from the hospital after 7 days, with no neurological deficit. Follow-up by conventional angiography 12 months after embolization showed complete obliteration of the aneurysm (Fig. 6). The patient suffered from transient mild headache, but no other neurological deficit in the 24-month clinical follow-up.

Discussion

Lenticulostriate aneurysms are rare, with a total of 23 cases reported so far in the literature. Etiological risk factors related to LSA aneurysm include hypertension, moyamoya disease, arteriovenous malformation, infection, systemic lupus erythematosus and substance abuse [2,5,6], although some cases were idiopathic. Clinical manifestations of LSA aneurysms are neurological signs such as headache due to meningeal irritation, stiff neck, and weakness or paralysis secondary to intracerebral hematoma, intraventricular hematoma or subarachnoid hemorrhage. Given the small number of cases reported in the literature, the natural history of LSA aneurysms has never been studied. However, in the cases reported thus far, follow-up after conservative management of ruptured LSA aneurysms revealed either spontaneous obliteration of the aneurysm or repeat rupture of the aneurysm, causing death [2]. The LSA aneurysm associated with moyamoya disease was reported to grow rapidly and the outcome of aneurysm rupture was poor [3].

Most reported cases of LSA aneurysms were treated surgically and, of the 16 that were surgically treated, nine required sacrifice of the feeding LSA, resulting in moderate disability in two cases [7]. The surgical challenges of LSA aneurysms include the fragile nature and extremely small size of the parent artery, and the deep location of the aneurysm. Endovascular management of LSA aneurysms has been reported in only two cases. Larrazabal et al. [4] embolized a ruptured LSA aneurysm and its parent artery with n-BCA. In that case, the n-BCA refluxed back into the MCA and led to moderate disability. Harrel et al. [3] occluded an LSA aneurysm and parent artery with no significant neurological complications, but the report lacked long-term follow-up data. In our present case, the aneurysm was obliterated while preserving the parent LSA, and no recurrence was noted after 1 year. The acute angle and small caliber of the parent vessels make endovascular access either difficult or impossible, thus leading to the potential need to sacrifice the parent artery. However, the advent of new microcatheters, guidewires and embolization agents allow the possibility of more endovascular treatments in the future.

In conclusion, the present case demonstrates that endovascular embolization of ruptured LSA aneurysms can be appropriate and effective. Indeed, treatment decisions of LSA aneurysm remain controversial only because of limited patient numbers and the uncertain natural history. Most reported cases of LSA aneurysms have been treated surgically. However, given the advances in microvascular technology and techniques, endovascular embolization of LSA aneurysm may be effective and more frequently applied in the future.

Conflict of interest statement

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

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