Case Report

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Endovascular Treatment of Common Carotid Artery Giant Aneurysm Using Intrasaccular Deployment Technique of Vascular Plug

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This case report describes a giant proximal common carotid artery aneurysm with partial thrombosis, which was treated by applying an Amplatzer vascular plug II (AVP II) for intrasaccular deployment and landing in the limited inflow zone. Endovascular embolization was suggested because of the high risk of open surgery and the difficulties of stenting. The patient passed the balloon occlusion test, and we attempted endovascular sacrifice of the carotid artery. The high flow status and limited inflow zone render such sacrificing challenging. Based on our experience in treating intracranial aneurysms, we used flow disruption intrasaccular Woven EndoBridge (WEB) to achieve embolization. The AVP II was designed similarly to the WEB device. We applied the same concept by deploying the AVP II intrasaccularly and landed in the aneurysm inflow zone. For a giant aneurysm with high flow and a limited landing zone, the AVP II can be deployed intrasaccularly by using a compressing technique to achieve complete occlusion of both the aneurysmal sac and the proximal inflow landing zone, thus preventing recanalization and endoleak.

Key words: common carotid artery, aneurysm, endovascular treatment, vascular plug, intrasaccular

Introduction

Extracranial carotid artery aneurysms (ECCAs) account for 0.4% of arterial aneurysms, and they are rarely located in the common carotid artery (CCA). Over the past 20 years, various treatments for ECCA have been developed; however, no preferred method or guideline has been established.[1] Many endovascular techniques have been reported for treating ECCAs. Considering the limitations of open surgical repair and the high risk of stroke and cranial nerve damage, more effective techniques are required.[2] In the present case, we used an Amplatzer vascular plug II (AVP II) to treat a giant aneurysm in the proximal CCA; the AVP II was applied to facilitate total embo-

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lization of the aneurysm, reduce recanalization, avoid the usage of coils for cost saving, and alleviate the mass effect to prevent cranial nerve compression. To the best of our knowledge, purposeful intrasaccular deployment of vascular plug to achieve complete embolization of aneurysm due to limited high flow landing zone had never been reported in the literature.

Case Report

A female patient aged 53 years presented with a neck pulsatile mass, dizziness, visual aura, and short-term loss of consciousness, but with no history of trauma. Computed tomography (CT) angiography revealed severe atherosclerosis in the left CCA and a 56 mm giant aneurysm with partial thrombosis at the proximal CCA (Fig. 1A & 1B). CT angiography and digital subtraction angiography revealed recanalization and enlargement of the sac associated with severe stenosis of the outflow zone (Fig. 1C). The patient tolerated clinical balloon test occlusion for 45 min and exhibited left hemisphere perfusion from the right internal carotid artery (ICA) via the anterior communicating artery and from the basilar artery via the posterior communicating artery (Fig. 1D & 1E). The cardiovascular specialist suggested endovascular treatment due to the high risk of open surgical repair and cranial nerve damage. Vascular diameter discrepancy, device diameter and working length limitations, low device radial force, poor vessel wall apposition, long-term usage of antiplatelet drugs, and risk of stroke were considered, and we concluded that

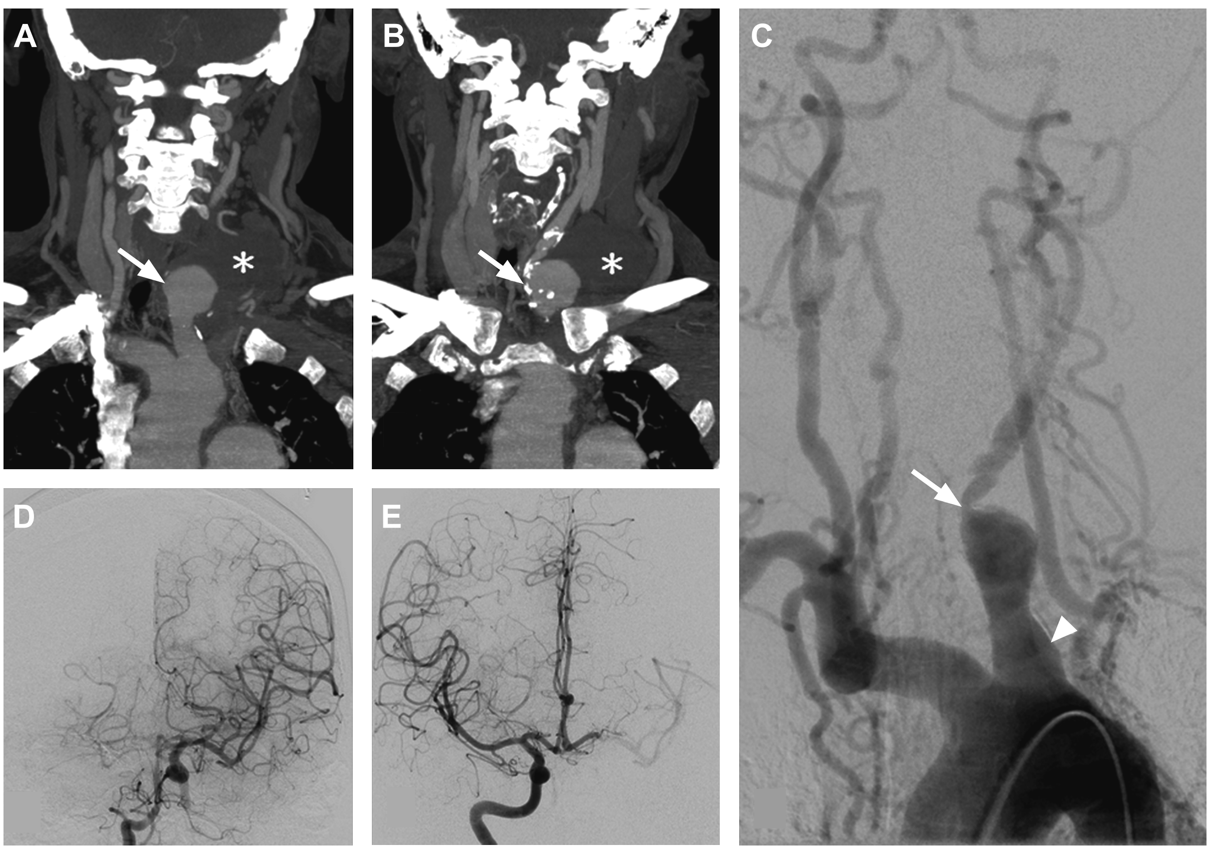

Fig. 1 Pre-embolization CT angiography, balloon occlusion test and aortography. (A and B) Giant proximal common carotid artery dissecting aneurysm with partial thrombosis (asterisk) and recanalized true aneurysm sac (arrows). Common carotid artery appeared severe atherosclerosis. (C) Aortography demonstrated left common carotid artery aneurysm with proximal dilatation (arrowhead) and distal severe stenosis (arrow). (D and E) Clinical balloon occlusion test. Good perfusion of the left hemispheric territory getting blood supply from right internal carotid artery via anterior communicating artery and from basilar artery via posterior communicating artery.

a covered stent was unsuitable. We performed endovascular sacrifice of the left carotid artery from the cervical ICA to the proximal CCA, including the aneurysm. Following bilateral puncture, a 7-Fr Shuttle sheath (Cook, Bloomington, IN) was placed in the proximal CCA, and an ENVOY 6-Fr distal access guiding catheter (Codman Neuro, Raynham, MA) with a coaxial Excelsior 1018 microcatheter (Stryker Neurovascular, Fremont, CA) was navigated across the stenosis to the ICA. The cervical ICA and distal CCA were embolized first to prevent an intraprocedural embolic shower. The challenges of sacrificing this giant aneurysm were extremely high flow status and limited safety stump from aortic arch; therefore, we placed Target XL Detachable Coils (Stryker Neurovascular, Fremont, CA) and intrasaccularly deployed an Amplatzer vascular plug II (AVP II, St. Jude Medical, Plymouth, MN), which was landed at the proximal CCA, to facilitate complete embolization and prevent recanalization (Fig. 2A). Aortography demonstrated total obliteration of the aneurysm and the proximal CCA without affecting the aortic arch flow (Fig. 2B). No procedure-related complications occurred. A 3-month follow-up CT angiography showed that the neck mass persisted with a reduced size without detectable recanalization. The previous transient ischemic stroke-like symptoms resolved completely (Fig. 3A). At 6-months follow-up, MR angiography revealed near-complete shrinkage of the aneurysm without recanalization (Fig. 3B), and the patient was free of clinical symptoms.

Discussion

ECCAs account for 0.4% of series involving the entire carotid entry, and they are rarely isolated from the CCA. These lesions pose high risk of neurological thromboembolism and cranial nerve compression, but they rarely rupture. The causes of ECCAs are unknown, but they are most commonly associated with atherosclerosis in older patients. Trauma, fibromuscular dysplasia, prior surgery or radiotherapy, congenital malformation, and infection can contribute to vascular degeneration and aneurysm formation.[3] ECCAs are divided into five types depending on the aneurysm location:[4] distal to the ICA (type 1), begin-

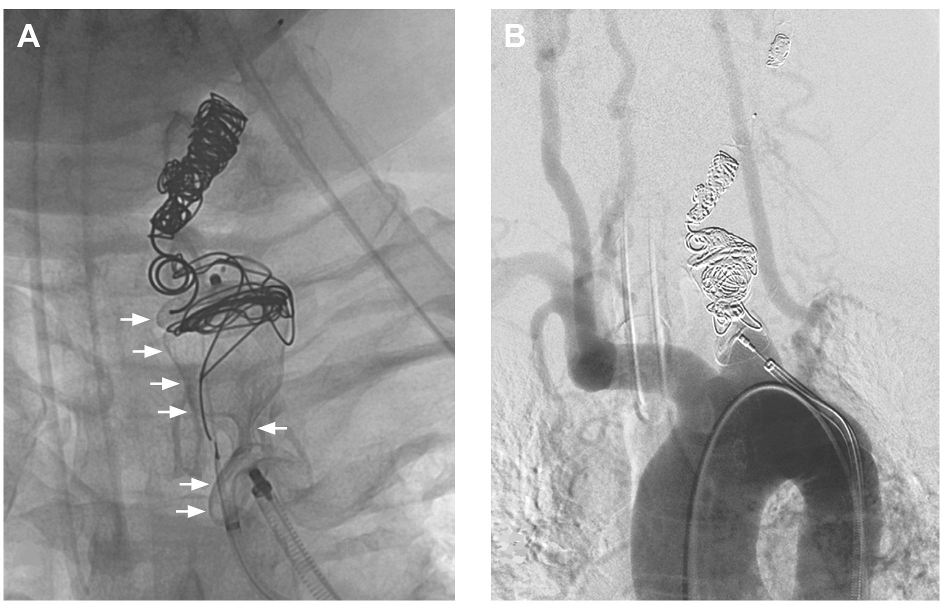

Fig. 2 Post-embolization fluoroscopy and aortography. (A) Intrasccular deployment of Amplatzer vascular plug II (arrows) for embolization of the aneurysm and the proximal dilated common carotid artery inflow zone. (B) Post embolization aortography revealed total obliteration of the aneurysm and proximal inflow common carotid artery.

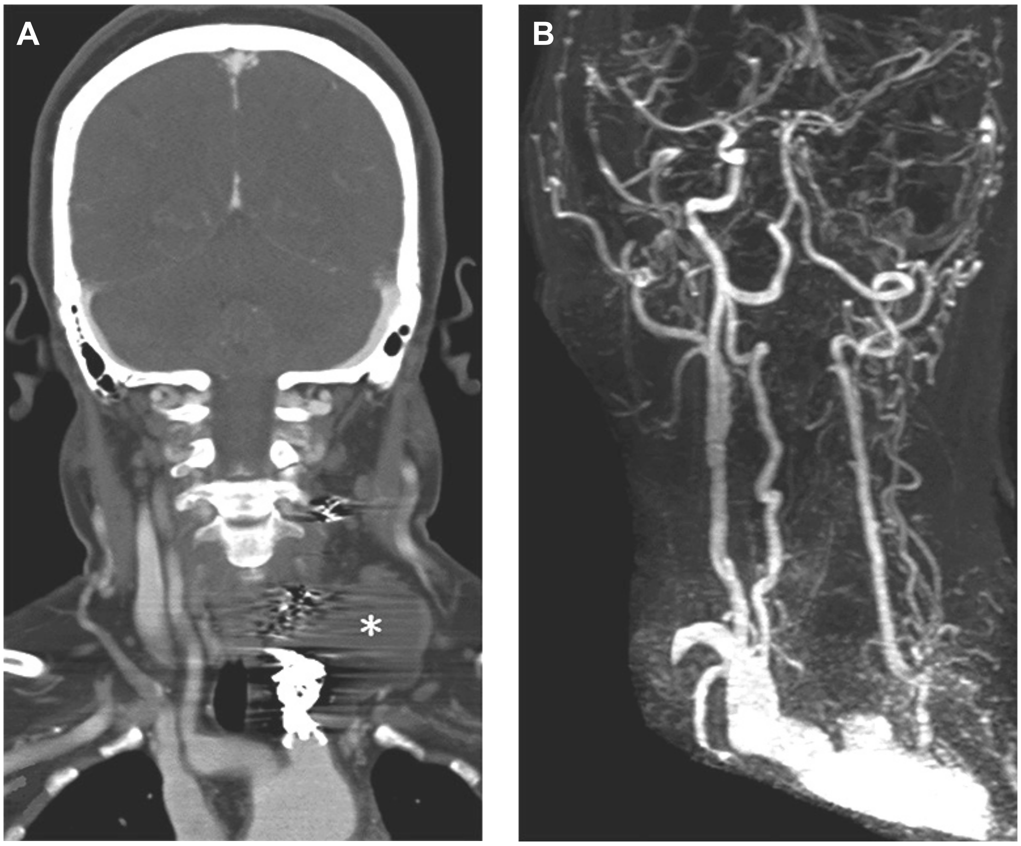

Fig. 3 Post-embolization imaging follow up. (A) The CT angiography at 3 months revealed decreased aneurysm size (asterisk) without recanalization. (B) The MR angiography at 6 months demonstrated nearly complete shrinkage of aneurysm without recanalization.

ning from the proximal ICA and extending toward the distal ICA (type 2), in the carotid bifurcation (type 3), in the ICA and CCA (long segmented aneurysms; type 4), and in the CCA (type 5). ECCA management is determined by the etiology, size, and location of the aneurysm. Sufficient experience and guidelines for choosing therapeutic options are lacking. Conservative medical treatment is acceptable for select patients with small asymptomatic aneurysms; however, a study concluded that conservative treatment may lead to stroke in > 50% of patients, with no treatment resulting in death in 60% – 70% of patients.[5] Open surgery comprising resection combined with reconstruction is the gold standard treatment, depending on the morphological classification of the aneurysm.[1] However, postoperative complications are frequent owing to invasiveness. The most common complication is cranial nerve damage, which occurs in 11% – 26% of cases.[4] Recently, advanced endovascular treatments such as covered stent placement, embolization, and stent-assisted coiling have become preferred management options because of their high success rate, reduced invasiveness, and low risk of cranial nerve injury.[6]

The important issue is while sacrificing the proximal CCA giant aneurysm. First, the partial thrombosis status of aneurysm may cause intraprocedual embolic shower and stroke. Second, it is difficult to achieve complete embolization via coils alone because of the extremely high flow status and limited length of proximal inflow CCA. As we know, intrasaccular embolization device designed to provide flow disruption for the intracranial aneurysm, and the Woven EndoBridge (WEB) device is used worldwide.[7] The AVP II contains unique tri-lobar segments and is made of a multi-layered densely braided woven nitinol mesh which generates six barrier planes for acceleration vascular occlusion. It can be shortened by compression, allowing better sealing and fitting for a short landing zone.[8] We placed some large Guglielmi detachable coils in the aneurysm to facilitate thrombosis, then we adopted the same concept of WEB device and deployed the first and second segments intrasaccularly; the third segment was landed at the limited inflow CCA zone. Conventional embolization of such large aneurysm at the proximal CCA requires numerous coils and has an extremely high recurrence rate due to high flow status and limited proximal embolized stump. The benefits of using the AVP II to manage a proximal CCA giant aneurysm include cost reduction, and to facilitate total embolization of the aneurysm, reduced recanalization, and reduced mass effect, which prevents cranial nerve compression. To the best of our knowledge, purposeful intrasaccular deployment of a vascular plug to achieve complete embolization of an aneurysm due to limited high flow landing zone had never been reported in the literature.

Conclusion

The Amplatzer vascular plug is indicated for the intraluminal occlusion of the feeding artery. For a giant aneurysm with high flow and a limited landing zone, the AVP II can be deployed intrasaccularly by using a compressing technique to achieve complete occlusion of both the aneurysmal sac and the proximal inflow landing zone, thus preventing recanalization and endoleak.

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