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Short Report

A Modified Technique of Open Surgical Treatment for Aneurysmal Sac Enlargement after Endovascular Repair

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ABSTRACT

Introduction: Although several articles have reported the successful treatment of an abdominal aortic aneurysm (AAA) enlargement after endovascular aortic repair (EVAR) due to endoleak or endotension, the strategy to treat this type of complication is still controversial.

Report: We report three cases of AAA expansion after EVAR. When other endovascular approaches were not considered effective, we performed a modified open surgical treatment.

Discussion: This technique includes ligation of all branched vessels arising from the aneurismal sac, plication of the aneurysm and wrapping of the aneurysm using bio-prosthetic material instead of explanting the implanted endovascular graft.

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Introduction

Endovascular intervention for the treatment of abdominal aortic aneurysm (AAA) represents one of the more recent standard treatment approaches. However, expansion of the AAA sac can cause persistent pressurisation of the aneurysmal sac after endovascular aortic repair (EVAR) and will require additional treatment. Although several articles have reported treatment for this type of complication, definite strategies are still controversial.^{1–3}

We present three cases of enlargement of the AAA sac after EVAR. Because additional endovascular repairs were not effective, we chose our modified open surgical treatments. The technique was feasible, but effectiveness can only be proven after long-term follow-up.

Report

Case 1: A 79-year-old man underwent EVAR of a 5.0-cm AAA, with the Zenith bifurcated endograft (Cook, Inc, Bloomington, IN, USA) (Fig. 1A1). However, computed tomography (CT) 4 years after the initial EVAR revealed that the aneurysmal size increased to 5.8 cm despite the lack of detectable endoleak (Fig. 1A2). Since the

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endoleak could not be detected by other imaging modalities, the patient underwent open surgery. His postoperative course was uneventful. Postoperative CT 6 months after the operation revealed the complete exclusion of AAA (Fig. 1A3).

Case 2: A 74-year-old man underwent EVAR of a 4.8 cm rapidly dilated AAA using the Excluder Endoprosthesis (W.L. Gore & Associates, Flagstaff, AZ, USA). Although the intra-operative angiogram revealed no endoleak, the postoperative CT showed type II endoleak (Fig. 1B1). Since CT 1 year after the initial EVAR still showed persistent type II endoleak and that the AAA diameter had increased to 5.3 cm, we performed coil embolisation of the responsible lumbar arteries. Although CT 4 years after the coil embolisation revealed no endoleak, the aneurysmal size had increased to 5.6 cm (Fig. 1B2). After the patient underwent open surgical treatment, CT 9 months after the surgery showed successful exclusion of AAA (Fig. 1B3).

Case 3: A 79-year-old woman underwent EVAR for a 4.2 cm rapidly dilated AAA with the Excluder Endoprosthesis. Although the postoperative CT showed type II endoleak, the primary cause was not clearly identified (Fig. 1C1). CT 4 years after the initial EVAR still revealed type II endoleak, and the aneurysmal sac increased to 5.3 cm in diameter (Fig. 1C2). Open surgery was performed, and CT 3 months after the surgery revealed the complete exclusion of the AAA (Fig. 1C3).

Technique: Written, informed consent was obtained from all patients prior to the procedure. After midline laparotomy, all the branched vessels, including the inferior mesenteric artery, lumbar arteries and the median sacral artery, were ligated. After the

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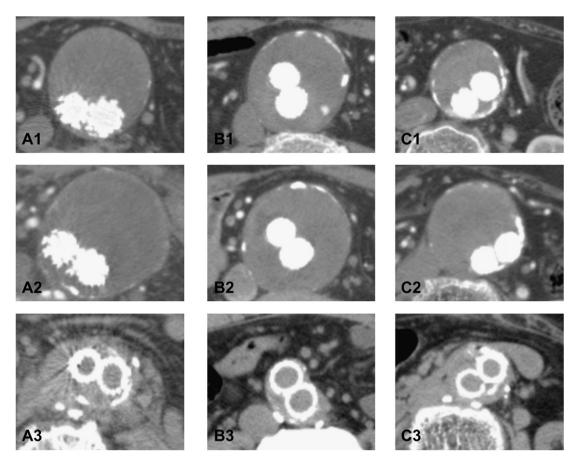


Figure 1. A; case 1. A1, no endoleak is detected on computed tomography (CT) after surgery. A2, CT 4 years after operation reveals no endoleak and increasing aneurysm size. A3, CT 6 months after open surgery shows completely repair of aneurysm. B; case 2. B1, postoperative CT showed type II endoleak. B2, although CT 4 years after the coil embolization revealed no endoleak, the aneurysmal size had increased. B3, CT 9 months after the surgery showed successful exclusion of aneurysm. C; case 3. C1, although the postoperative CT showed type II endoleak, the primary cause was not clearly identified. C2, CT 4 years after the initial revealed the aneurysmal sac increased. C3, CT 3 months after the surgery revealed the complete exclusion of the aneurysm.

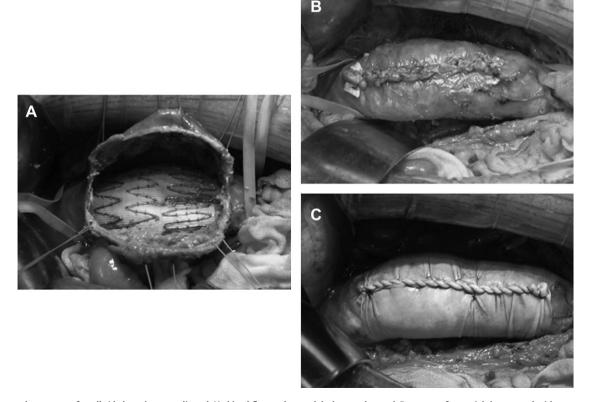


Figure 2. A, opened aneurysm after all side branches were ligated. No blood flow and no endoleak were detected. B, stent-graft was tightly wrapped with aneurysm wall. C, the aneurysm wall was additionally wrapped with Xenomedica patch.

aneurysm was completely exposed, the aneurysmal sac was longitudinally opened. Subsequently, we confirmed that no blood flowed from the orifices of branched arteries, as well as the absence of type I and III endoleaks. The anterior surface of the aneurysmal wall was partially resected to eliminate the dead space between the endograft and the aneurysmal wall. Thereafter, the endograft was tightly wrapped with the aneurysmal wall by a running suture. Finally, a Teflon-backed glutaraldehyde-preserved equine pericardium (Xenomedica; Baxter Healthcare Corp, Horw, Switzerland) was wrapped and sutured around the aneurysmal sac (Fig. 2). The retroperitoneum was closed to avoid aorto-enteral fistulae.

Discussion

An enlargement of AAA after EVAR may be found, and our strategy for the treatment of an expansion due to type II endoleak or endotension is as follows. We initially perform the retrograde coil embolisation for type II endoleak. However, if the treatment is not considered to be effective, or the culprit vessels of type II endoleak are unclear with the use of imaging modalities or any endoleak is undetected, open surgical repair is considered to be a good indication. As an open repair, combined graft explantation followed by the conventional graft replacement and aortobiiliac bypass grafting can be feasible treatments of choice. However, these procedures present some challenges because of difficulties associated with aortic cross-clamp and complete graft explantation.⁴ Therefore, we designed this modified technique in an attempt to avoid aortic clamp, graft explantation and systemic heparinisation. Ferrari et al. reported that similar cases with unsolved type II endoleaks could be fixed with surgical sacotomy and ligation of the patent backbleeding vessels.⁵ However, we think that newly occurred endotension or seroma cannot not be prevented by only this procedure. In the case with a large aneurysm, we can achieve plication and wrapping of the aneurysm after the aneurysmal wall is resected as much as possible, but the exposure of the backside of the aneurysm might be risky. As long-term follow up is crucial to determine the benefit of respective interventions, a limitation is the short follow-up of the cases.

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Conflict of Interest

None.

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