Successful hybrid management of a ruptured abdominal aortic aneurysm induced by type II and IIIB endoleaks after endovascular aortic repair

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ABSTRACT
Ruptured abdominal aortic aneurysm after endovascular abdominal aortic repair is a relatively rare condition. The management of this type of a rupture is challenging and controversial. We report here a case of ruptured abdominal aortic aneurysm 6 months after endovascular abdominal aortic repair. Although the main cause of this rupture was initially believed to be a type II endoleak, it was also a type IIIB endoleak practically. The patient was successfully treated via the hybrid approach. He recovered well, with no endoleaks for the next 6 months. (J Vasc Surg Cases and Innovative Techniques 2019;5:323-6.)

Keywords: Endovascular abdominal aortic repair; Abdominal aortic aneurysm rupture; Type II endoleak; Type IIIB endoleak

Late rupture of an abdominal aortic aneurysm (AAA) after endovascular abdominal aortic repair (EVAR) is a relatively rare condition, with a reported incidence of 0.9%. The predominant cause of such a rupture is a type I or III endoleak, whereas AAA rupture owing to a type II endoleak is rare. The management of this type of AAA rupture is challenging, and the optimal use of several treatment approaches, including surgical or endovascular approaches, remain debatable.

Here, we describe a case of AAA rupture after EVAR owing to type II and IIIB endoleaks, which was successfully treated via a hybrid approach that combined inferior mesenteric artery (IMA) ligation and placement of a new endograft within the previous graft. The patient’s consent was obtained for this case report.

CASE REPORT
A 74-year-old man with chronic renal failure and alcoholic cirrhosis underwent EVAR for the treatment of a 50-mm AAA with implantation of the Endurant II (25-× 16-× 145-mm bifurcated endograft, 16-× 20-× 124-mm contralateral limb to the left, and 20-× 20-× 82-mm iliac extension to the right; Medtronic Endovascular, Santa Rosa, Calif). Completion angiography after the EVAR procedure detected a type IV endoleak (Fig 1), and the patient was monitored by computed tomography (CT) scans during the follow-up period in an outpatient clinic. CT imaging 3 months after the EVAR revealed a slight enlargement of the AAA by 1 mm, and the next CT imaging was scheduled after 3 months later. About 6 months after the EVAR, he experienced a sudden onset of abdominal pain during endoscopy at another hospital and it gradually got worse. CT imaging revealed an enlarged AAA and a retroperitoneal hematoma, and a ruptured AAA was diagnosed. The patient was transferred to our hospital.

CT angiography revealed slight contrast filling in the aneurysm sac in the arterial phase and an obvious filling in the delayed phase (Fig 2). Although a type II or III endoleak was suspected, it
was difficult to specifically identify the endoleak type. Hence, the patient was taken to the hybrid operating room, where both the endovascular and surgical procedures could be performed.

The first angiography to determine the location of the endoleak revealed a type II endoleak from the IMA (Fig 3). Owing to the emergent and unstable nature of the aneurysm, we performed a laparotomy to ligate the IMA, and a repeat angiography showed no obvious endoleak (Fig 4). However, considering the possible involvement of other drainage vessels and because it was considered secure to perform sacotomy without clamping the proximal site of the AAA because of no obvious endoleak, we decided to perform sacotomy to assess these vessels.

After sacotomy and clot removal, no retrograde bleeding was recorded from any of the arterial branches. However, two sites of active blood leakage from the main body and the leg of the endograft and blood oozing from the endograft (Fig 5) were noted, confirming a type IIIB endoleak. The sites of active blood leakage were nearly corresponding with the location of the endoleak at preoperative CT angiography. Considering our patient’s comorbidity, explantation of endograft and graft replacement was believed to be extremely invasive; therefore, a less invasive approach of the endovascular procedure was selected. A 23 × 3-mm Excluder Aortic Extender Endoprostheses (W. L. Gore & Associates, Flagstaff, Ariz) was deployed within the main body of the previous endograft, and 20 × 10-mm and 18 × 10-mm Excluder Contralateral Leg Endoprosthesis (W. L. Gore & Associates) were deployed in each leg of the previous endograft; the disappearance of the type IIIB endoleak was confirmed by gross observation. The procedure was completed after plicating the aneurysm sac.

Contrast-enhanced CT 6 months after this procedure revealed no endoleak, and the peritoneal hematoma was found to be completely resorbed. Although the patient was forced to begin artificial dialysis, he was ultimately discharged.

**DISCUSSION**

EVAR has gained popularity as a widely used alternative to the conventional surgical repair of AAA since Parodi et al first reported about EVAR for the treatment of AAA in 1991. Although EVAR leads to less perioperative mortality and morbidity as compared with the conventional surgical repair approaches, endoleak after EVAR remains a troublesome complication, and the fate of the endoleak is unclear.

Type II endoleak is the most common complication, and its management remains controversial. Type II endoleak occurs because of retrograde blood flow into the aneurysm sac, mostly from the IMA or a lumbar artery. Type II endoleaks may be divided into two types: type IIA and type IIB. Type IIA endoleaks result from a single causative vessel that originates from the aneurysm sac with a to-and-fro flow, and it tends to resolve spontaneously. In contrast, a type IIB endoleak involves multiple vessels that can become inflow vessels or drainage vessels, respectively. Type IIB endoleaks can persist, and, consequently, enlarge and pressurize the aneurysm sac, thereby increasing the risk of rupture. Paradoxically, in cases of AAA rupture owing to a type IIB endoleak,
even if the culprit inflow vessel has been embolized, it is believed that some drainage vessels remain, which function as new inflow vessels.

A type III endoleak after EVAR has a considerably higher risk of potential aneurysm rupture, often requiring reintervention.6 A type III endoleaks can also be divided into two types; a type IIIA endoleak originates from disconnections in the endografts, whereas a type IIIB endoleak originates from a defect in the endograft. It can be tough to detect a type IIIB endoleak by angiography or CT angiography, and it can mimic a type II endoleak on CT angiography.7 Similar to that in our case, it is often detected only during open surgery. Although it remains unclear whether a type IIIB endoleak can enlarge the aneurysm sac, the cause of the endoleak needs to be recognized when the type cannot be accurately identified.7 Multiple approaches for the management of type IIIB endoleaks have been proposed.7-9 For instance, if the anatomic condition is amenable, an endovascular approach is less invasive than explantation of the endograft and graft replacement.

Late rupture of AAA after EVAR is extremely rare, with a reported occurrence of less than 1%.1 Type I and III endoleaks are the predominant causes of AAA rupture, and delayed rupture owing to type II endoleaks are rare. Careful attention must, however, be paid in selecting the appropriate procedure for ruptured AAA repair after EVAR because of the presence of coagulation disorders and peritoneal hematoma; these conditions differ from those expected with elective reintervention for a nonruptured AAA. Most cases of late rupture of AAA after EVAR owing to type II endoleaks are categorized as type IIIB endoleaks,5 which implies that multiple vessels are involved. Hence, even if a single causative vessel is ligated or embolized, other drainage vessels may turn into new inflow vessels, leading to sustained bleeding from the aneurysm sac. Furthermore, considering that a type IIIB endoleak is difficult to diagnose without sacotomy, it is essential to perform sacotomy to ensure there is no blood flow into the aneurysm sac in cases of ruptured AAA after EVAR. In case of ruptured AAA induced by type II and IIIB endoleaks, such as in our case, it is difficult to determine the best approach for management. Explantation of the endograft and graft replacement are definitive management steps, albeit extremely invasive. If the anatomic condition is amenable, a hybrid approach that combines causative artery ligation and EVAR, like in our case, is less invasive and an alternative management approach.

It has been reported that both isolated type II and type IIIB endoleaks lead to late rupture of AAA8,10; therefore, it was difficult for us to determine the main cause of AAA rupture in our case. Because only the IMA was the causative artery for the type II endoleak in our case, this endoleak was categorized as a type II A; however, our patient’s type II endoleak sustained. It was thus speculated that the type IIIB endoleak acted as an inflow vessel, and the type II endoleak sustained like a type IIIB endoleak; consequently, our patient’s AAA enlarged and ruptured.

CONCLUSIONS

We have described the successful application of a hybrid approach to treat a ruptured AAA after EVAR, which is induced by type II and IIIB endoleaks. Our
case description suggests that, even in cases of ruptured AAA after EVAR, whose main cause is believed to be a type II endoleak, the presence of other drainage vessels or an unrecognized type IIIb endoleak could result in sustained bleeding.

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