

# Ruptured Abdominal Aortic Aneurysm with Antecedent Endovascular Repair of Abdominal Aortic Aneurysm

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Late aortic rupture following successful endovascular repair of abdominal aortic aneurysm still does occur. It represents the ultimate failure of endovascular aortic repair of abdominal aortic aneurysm (EVAR) and subjects patients to equivalent risk of death as de novo rupture. Unfortunately, it is difficult to identify patients at risk for post-EVAR rupture as many present with aortic rupture in the absence of any endograft-related complications. Continued surveillance and timely intervention are of paramount importance to assure rupture-free survival, the ultimate goal of any aneurysm treatment modality. The vascular surgeon needs to be prepared to provide the optimal therapy, whether open or endovascular, for this challenging cohort of patients.

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## INTRODUCTION

Despite continuously expanding applicability with its superior outcomes compared with open repair (OR) [1-3], endovascular aortic repair of abdominal aortic aneurysm (EVAR) is not completely effective in reaching its ultimate goal: prevention of abdominal aortic aneurysm (AAA) rupture and death. AAA sac expansion and rupture after technically successful EVARs do occur, and it remains a major concern as not only it represents a failure of treatment but causes high mortality rate.

The vascular surgeon needs to be aware of, understand this entity and be equipped to treat patients with ruptured AAA (rAAA) with history of EVAR. This article reviews the disease process and therapeutic options.

## INCIDENCE, RISK FACTORS

The incidence of late aneurysm rupture after EVAR

is difficult to assess because many are case reports, and many derive from subgroups of patients treated with specific devices that are no longer in use [4]. In a recent meta-analysis of published randomized controlled trials and large databases (Medicare and SwedVasc databases), a significantly higher rate of rAAA was noted after EVAR [5]. Later rupture of aneurysm occurs due to incomplete exclusion of the aneurysm from circulation by a variety of endoleaks and endotension. The reported incidence of incomplete exclusion ranges from 6% to 50% [6], while the cumulative annual risk of rupture after EVAR varies from 0.5% to 1.2% per patient per year after EVAR [6-9]. Wyss et al. [10] reported a total of 27 post-EVAR rAAAs during a mean follow-up of 4.8 years in EVAR trials (0.8 ruptures per 100 person-years). Five (18.5%) ruptures occurred within 30 days of EVAR; three of which occurred in-hospital. The remaining 22 patients presented with ruptures more than 30 days after EVAR.

Predictors of late AAA rupture after EVAR have been

identified. With respect to aortic anatomical features before intervention, larger aneurysm diameter seems to be associated with an increased risk of graft rupture [6-9,11-13]. Not only is the diameter the primary determinant of the risk of primary aneurysm rupture, but also a strong predictor of late ruptures, type I endoleak and aneurysm-related death. Zarins et al. [13] in a review of 923 patients treated with AneuRx stent graft found that preoperative AAA  $\geq 6.0$  cm was the only independent predictor of the aneurysm-specific end point of rupture, AAA growth, AAA-related death and conversion. Peppelenbosch et al. [9] in a survey of data from 4,392 patients in the European Collaborators on Stent-Graft Techniques for Abdominal Aortic Aneurysm Repair (EUROSTAR) registry, showed that the ratio of aneurysm-related to unrelated death was about 50% in patients with AAA  $\geq 6.5$  cm compared with 28% in a smaller aneurysm group. The mean preoperative AAA size of those that ruptured after EVAR is 6.5 cm in Lifeline registry [13].

Post procedure factors include type I, II, and III endoleaks [4-19], graft migration [8,20,21], kinking [8], sac growth [9] and poor compliance with follow-up [22]. Of these, endoleak is the main culprit [7,20,23]. It is of interest however to note that in the EVAR trials, while 17 (63%) of the 22 late ruptures had history of complications or signs of failed EVAR more than 30 days after the repair, nearly 20% (5/22) of the ruptures had no history of endograft-related complications or signs of failure [24]. A series from University of Pittsburgh showed 77% of patients showed no evidence of endoleak at last follow-up [25]. Similarly, in the AneuRx trial, 5 of 7 patients with post-EVAR ruptures showed no evidence of endoleak at last follow-up before rupture [13]. It has been shown that up to 40% of patients had no abnormalities detected at last follow-up before rupture [7,20]. The absence of endoleak during follow-up does not guarantee protection rupture.

Stable AAA sac size does not assure one of cure of the aneurysm [22,23]. Only a small percentage of post-EVAR rAAA had shown sac growth at last follow-up before rupture [25]. Furthermore, AAA sac that has been shrinking may re-expand over time [26]. It was also noted that the majority of endoleaks found at the time of rAAA were new and 77% of patients showed no evidence of endoleak at last follow-up in this study. Others have also noted that up to 40% of patients had no abnormalities detected at last follow-up before rupture [7,20]. The absence of endoleak during follow-up does not mean that all is well, and continued surveillance after EVAR cannot be overemphasized.

## TREATMENT

Therapeutic approach to post-EVAR rAAA is dependent on the anatomic characteristics and the culprit lesion. While it is ideal to remove all the endograft to eliminate any possible recurrent problems [27], complete excision is not an easy task. A variety of issues may complicate the matter and include: barbs or hooks; suprarenal fixation stents or Palmaz stents that are well-incorporated into the aorta; inflammatory changes around the aorta and vena cava, left renal vein and iliac veins all elevate the complexity of the operation. Complete excision of suprarenal fixation endograft, especially with well-adhered Palmaz stent, may leave the aortic or arterial wall too thin and denuded, increasing the risk of anastomotic tear. Recent iteration endografts with aggressive suprarenal fixation system may present increased risk of complications. In such cases, partial resection of endograft may significantly reduce the risk of reno-visceral ischemia time and adverse outcomes. The endograft remnant can be incorporated in the suture line along with the aortic wall it is sewn to the surgical graft [27-29]. Complete excision would be required in the setting of graft infection, however.

When total excision of endograft with suprarenal fixation system and/or Palmaz stent across the renal orifices is required, retroperitoneal approach with aortotomy along the lateral wall allows the surgeon improved exposure, and ability to inspect the luminal surface of the aorta after removal of the devices and repair the defects, if necessary. The aortotomy may then be closed primarily to the level of the renal arteries and then the surgical graft may be sewn at that level.

Often, endovascular solution is a viable option for a post-EVAR aortic rupture. Careful review of the computed tomographic angiography is essential to identify the etiology of rupture and for proper preoperative planning. A large sheath (12- or 14-French, 45 cm long) is placed over a super-stiff wire and positioned at the level of the renal artery. This allows placement of the compliant aortic occlusion balloon into the supraceliac aorta to achieve hemostasis, should that become necessary. Contralateral artery access is also required to place a marker pigtail catheter.

Once arteriography reveals the culprit lesion, the aortic occlusion balloon is removed and the extension graft is inserted through that side, if patient's hemodynamic condition permits. If that is not possible, the pigtail catheter is removed and the extension graft is inserted through that side. Aortogram is then obtained through the large sheath that is supporting the aortic occlusion balloon. Once the target anatomy is confirmed, the balloon is deflated and withdrawn, followed by deployment of the extension graft.

## OUTCOMES

The reported operative mortality rates for post-EVAR rAAA range widely from 15% to 67% [7,16,20,23,28-33]. May et al. [16] observed a significantly lower mortality rate of 17% with OR of post-EVAR rAAA compared with 54% in patients with de novo rAAA, and postulated its survival benefit to relative hemodynamic stability. This finding has been corroborated by several investigators. Coppi et al. [23] found that hemodynamic instability was the only predictor of death in their comparative study of 14 post-EVAR rAAA with 155 de novo rAAA; however, despite the lower frequency of hemodynamic instability in post-EVAR rAAA, similar 30-day mortality rates were found between the two groups (28.5% vs. 38.7%, respectively). Mehta et al. [28] in a series of 27 post-EVAR rAAAs, of whom 26 underwent treatment, noted that hemodynamic instability was observed in only 19% of their patients and reported a 15% operative mortality rate (4/26).

Most studies, however, report significantly higher mortality rates with repair of post-EVAR rAAAs. In a review of 270 late AAA ruptures after EVAR, Schlösser et al. [7] reported a 43% (69/164) operative mortality rate. Fransen et al. [20] also reported from EUROSTAR registry data a 62% operative mortality rate in 34 post-EVAR. Wyss et al. [10] observed from EVAR trials a 67% (18/27) 30-day

mortality rate. Cho et al. [25] reported in a University of Pittsburgh series of 18 delayed rAAAs after EVAR and 233 de novo rAAAs nearly identical in-hospital mortality rates (39% vs. 37%); the frequencies of hemodynamic instability were similar (56% vs. 53%) as were the frequencies of preoperative cardiopulmonary resuscitation (17% vs. 22%).

## CONCLUSION

Post-EVAR aortic aneurysm rupture does occur and is as lethal as de novo types. The presence of endograft does not necessarily confer survival benefits when the aneurysm ruptures. Sac shrinkage or the lack of endograft-related complications, such as endoleak, does not necessarily eliminate the risk of rupture. Thus, continued surveillance is mandatory for the remaining life span of these patients, even in the absence of any complications. An aggressive re-intervention program for treatment of complications associated with sac expansion may help reduce this late rupture rate. With widespread technology and long-term follow-up post-EVAR rAAA are expected to increase. OR may add an additional layer of complexity in the setting of suprarenal fixation and fenestrated endografts. The vascular surgeon should be well equipped to manage this highly lethal condition.

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