# Treatment of Abdominal Aortic Pathology

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**KEYWORDS**

• Abdominal aortic pathology • Endovascular repair • Abdominal aortic aneurysm • Treatment

**KEY POINTS**

- There are many treatment options for abdominal aortic pathology, particularly with the ever-expanding endovascular techniques.
- Surgical intervention is recommended when there is rapid aneurysmal expansion or the development of symptoms such as abdominal pain, tenderness, and back pain.
- Randomized, controlled studies and long-term results have shown better short- and medium-term outcomes when comparing endovascular aneurysm repair with open repair.
- Open aortic repair is increasingly being reserved for those with complex anatomy or a coexisting disease process that prohibits them from an endovascular repair.
- Current guidelines recommend endovascular repair for Trans-Atlantic Society Consensus (TASC) A disease and open repair for TASC D disease; there is an increasing trend for TASC B, C, and D lesions to be treated endovascularly.

## INTRODUCTION

Abdominal aortic pathology is a diverse topic, ranging through a broad span of possible pathologies. The treatment options are equally vast, particularly with the ever-expanding endovascular techniques. In this article, we discuss management strategies for abdominal aortic aneurysms (AAA) and aortic occlusive disease, because they represent some of the most common pathologies encountered in clinical scenarios.

## ANEURYSMAL DISEASE

Arterial aneurysms are defined as dilation greater than 1.5 times the expected arterial size.[1] In the abdominal aorta, the expected diameter is 2 cm; therefore, dilation greater than 3 cm is considered aneurysmal. Aortic aneurysms typically occur in the infrarenal aorta, but can occur anywhere along the vessel. The prevalence of AAA increases with age and is more common in men, occurring in 7% to 8% of men over 65 years of age.[2–5] Other etiologic factors include smoking, ethnic origin, family history, hypercholesterolemia, hypertension, and prior vascular disease.[6] Although aneurysms do increase with age, there are multiple processes that can contribute to aneurysm formation at younger ages, including inflammatory, infectious, genetic, and traumatic etiologies.

### *Indications for Elective Therapy*

Surgical intervention is recommended when there is rapid aneurysmal expansion (>1 cm/y) or the development of symptoms such as abdominal pain, tenderness, and back pain, regardless of size. Such patients are at a higher risk of rupture as compared with asymptomatic patients or those with slower growth.[7,8] Treatment is also recommended when max diameter reaches 5.0 to 5.5 cm, owing to the relative risk of mortality associated with repair compared with aneurysm rupture.[9,10]

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Cardiol Clin ■ (2017) ■–■
http://dx.doi.org/10.1016/j.ccl.2017.03.009

### *Endovascular Treatment*

Multiple, high-quality, randomized, controlled studies have shown better short- and medium-term outcomes when comparing endovascular aneurysm repair (EVAR) with open repair. Additionally, long-term results have been shown to be at least equivalent.[11–14] These data have driven EVAR to become the primary mode of therapy for the majority of patients with AAA. Open repair is generally preserved for patients with complex anatomy and is discussed elsewhere in this article.

With the advent of increasingly complex endovascular techniques and expanding indications for use, there has been an increase in use for such devices and both Medicare and the National Inpatient Sample have seen a decrease in the number of open aneurysm repairs performed.[11,12] There are a variety of endografts available on the market today and endovascular therapy represents approximately 70% of aortic aneurysm therapy provided today. New clinical trials and off label use of various devices has resulted in escalating innovation within endovascular therapy.

Although EVAR is an attractive option for an increasing number of patients, there are limitations to its use. The EVAR trial 1 and 2 (UK Endovascular Aneurysm Repair Trial 1 and 2) was a randomized control trial that looked at EVAR versus no intervention in high-risk patients with aneurysms greater than 5.5 cm. Although the 30-day mortality results were higher in the EVAR group, they demonstrated no difference between aneurysm-related or overall mortality between groups at 4 years. They also concluded that very high-risk patients do not benefit from AAA repair because they die from other causes before a benefit can be realized.[13,15] Despite this finding, many groups advocate for use of EVAR even in high-risk patients. Data from the VA National Surgical Quality Improvement Program showed that high-risk veterans undergoing EVAR showed a 30-day mortality rate of 3.4% and a 1-year mortality rate of 9.5% (**Table 1**).[16]

**Table 1**
**Results of major open versus EVAR trials**

| | Cardiac Morbidity (%) | | All-Cause Mortality (%) | |
|---|---|---|---|---|
| | Open | EVAR | Open | EVAR |
| DREAM | 5.7 | 5.3 | 4.6 | 1.2 |
| OVER | — | — | 4.7 | 1.7 |
| EVAR-1 | 2.7 | 1.4 | 3.0 | 0.5 |

*Abbreviations:* DREAM, Dutch Randomized Endovascular Aneurysm Management; EVAR, endovascular aneurysm repair; OVER, Standard Open Surgery Versus Endovascular Repair of Abdominal Aortic Aneurysm.

### *Postoperative complications*

There are not a suitable amount of data to properly compare differences and complications between difference devices, given the wide variety and indications for use of different devices and the lack of long-term follow-up.

**Endoleak** An endoleak is defined as persistent blood flow within the aneurysm sac after EVAR. There are 5 recognized categories of endoleak, with distinct etiologies and treatment strategies.

- *Type I endoleak*: A type 1 endoleak describes blood flow from around either the proximal (type 1a) or distal (type 1b) seal of the endograft. Type 1a endoleaks are typically treated in the operating room by deploying an extension or bare metal stent around the area of the leak. Type 1b endoleaks within the common iliac artery are treated by distal extension of the graft within the external iliac artery and occlusion of the hypogastric artery. When type 1 endoleaks are not responsive to traditional treatment strategies, an operative explant of the graft should be attempted owing to the high rate of rupture associated with type 1 endoleak.
- *Type 2 endoleak*: A type 2 endoleak is caused by backbleeding aortic branches into the aneurysm sac. The inferior mesenteric artery, lumbar arteries, or the middle sacral artery are responsible for such a leak. Of treated patients, 10% to 20% have evidence of type 2 endoleaks on postoperative computed tomography (CT) scans after EVAR.[17–20] Up to 80% of these leaks resolve within 12 months after EVAR.[21] Type 2 endoleaks are not treated unless there is enlargement of aneurysm sac size as because is a low likelihood of rupture with type 2 endoleaks.[20,22] Techniques to exclude type 2 endoleaks involve excluding the inflow and outflow vessels into the aneurysm sac.
- *Type 3 endoleak*: Type 3 endoleaks occur when there is destruction to the endograft's fabric or a leak between components of the graft. This can occur when there is stent graft fatigue in which the barbs or struts of the stent fracture under the continual mechanical stress during the cardiac cycle. Type 3 endoleaks occur in 0% to 1.5% of EVARs.[20,23] They can be successfully treated by relining the graft.
- *Type 4 endoleak*: Type 4 endoleaks are caused by porosity of the endograft. They