Treatment of Complications Following Endovascular Repair of Abdominal Aortic Aneurysms

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ABSTRACT

Endovascular aneurysm repair (EVAR) is an important treatment option for abdominal aortic aneurysms, with lower perioperative morbidity and mortality rates than open surgical aneurysm repair. However, EVAR is associated with several unique complications that are not encountered with surgical repair such as endoleaks, graft migration, and renal artery occlusion. Preservation of the morbidity and mortality advantages of EVAR relies on the successful treatment of these complications by minimally invasive, endovascular approaches. Some of the techniques used to treat EVAR complications include balloon dilation and stenting, deployment of additional stent-graft pieces, coil embolization, and thrombolysis. Although the employment of these endovascular salvage techniques is common, data regarding their intermediate- to long-term efficacy is sparse, and further studies are needed to determine their efficacy in preventing conversion to open aneurysm repair and aneurysm rupture.

KEYWORDS: Endovascular repair, aortic aneurysm, stent graft, endoleak

Objectives: On completion of this article, the reader should understand the major complications of endovascular abdominal aortic aneurysm repair, as well as the methods by which these complications are diagnosed and treated.

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Endovascular repair for infrarenal abdominal aortic aneurysms (AAAs) was introduced into clinical practice by Juan Parodi and associates in 1991.¹ Initially, this procedure was reserved for patients who were deemed too high risk for traditional open aneurysm repair. However, increasing interest in this procedure among both patients and physicians has led to the use of

this technology in patients who otherwise would have been suitable to undergo a traditional open aneurysm repair. One factor in the increasing utilization of the endovascular aneurysm repair (EVAR) procedure is the improvement in device designs, which has expanded the number of patients in whom endovascular repair is anatomically feasible. An additional reason for the

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increasing use of EVAR is the accumulating evidence from large case series and recently from randomized controlled clinical trials showing that patients treated with EVAR have significantly lower mortality and systemic morbidity rates in the perioperative period as compared with patients treated with transperitoneal or retroperitoneal surgical repair.^{2,3} In fact, the less physiologically stressful EVAR procedure has been shown to be superior to open repair in regard to a multitude of perioperative parameters such as length of operation, operative blood loss, transfusion requirements, and length of intensive care unit and hospital stay.^{2,3}

Although EVAR represents an important addition to the treatment options for AAAs, enthusiasm for the procedure should be tempered by the unique complications of this procedure and by the lack of data on the long-term outcomes of EVAR compared with open AAA repair. The EUROSTAR collaboration has published intermediate-term follow-up data from their large case series that showed that patients treated with EVAR continued to experience aneurysm rupture at a rate of ~1% per year. An additional 2.1% of patients required conversion from an endovascular graft to a conventional graft each year, a procedure that has significantly higher morbidity and mortality rates than primary open AAA repair.^{4,5} Although perioperative systemic complications occur at lower rates after EVAR when compared with open repair, local and graft-related complications are more frequent in patients treated with EVAR. In addition, endovascular grafts are susceptible to additional complications that do not occur with traditional grafts, such as endoleaks and graft migration. These occur because the endovascular technique neither utilizes suture material to hold the stent graft in place nor ligates the visceral arteries that insert into the aneurysmal aorta.^{3,6-8}

Secondary procedures to treat graft-related complications are also necessary more frequently after endovascular repair. Secondary procedures may utilize endovascular or open surgical techniques.⁹⁻¹¹ The EUROSTAR registry midterm data reveals that 18% of patients who underwent EVAR needed a secondary procedure after initial graft placement, at a mean of 14 months after the procedure. The freedom from intervention rate at 4 years, the longest follow-up subgroup, was 62%. Overall 76% of the secondary procedures were endovascular, but the remaining 23% were either transperitoneal repairs or extra-anatomic bypass procedures.¹² Other large cases series have confirmed the high rate of secondary interventions necessary after EVAR, with reintervention rates ranging from 12 to 28%.¹⁰ To date, no large scale randomly controlled trial has released data comparing long-term patient outcomes, complications, and reintervention rates of EVAR and open aneurysm repair techniques.

This article will review the systemic and graft-related complications that occur after endovascular

aneurysm repair. The methods by which these complications are treated will also be examined, with a particular focus on endovascular techniques.

COMPLICATIONS OF EVAR

The Ad Hoc committee for Standardized Reporting Practices in Vascular Surgery of The Society for Vascular Surgery/American Association of Vascular Surgery classifies complications as deployment-related, implant-related, or systemic in nature. In addition, complications are graded by severity based on the clinical sequelae and the invasiveness of the procedures necessary to treat the complication.¹³ Complications related to device deployment include operative bleeding, aortic dissection, arterial perforation, peripheral embolization, formation of an access site hematoma or pseudoaneurysm, and access site infection. Implant-related complications include aneurysm rupture, device migration, device infection, endoleak, aortoenteric fistulae, limb thrombosis or occlusion, and buttock or leg ischemia or claudication. Several cases series have reported higher access- and graft-related complication rates in patients whose aneurysms were treated using endovascular techniques.^{7,13}

The higher rates of local complications associated with EVAR are countered by lower rates of systemic complications in these patients. Systemic complications include damage to the cardiac, pulmonary, renal, cerebrovascular, gastrointestinal, and hematologic organ systems.^{8,9} In addition, a substantial fraction of patients develop "postimplantation syndrome," which consists of fever, leukocytosis, elevated C-reactive protein, and local abdominal tenderness in the absence of demonstrable infection.^{7,14,15} The reduction in cardiac complications results from the decreased physiological stress of endovascular repair resulting from the reduction of intraoperative blood loss, the elimination of aortic cross-clamping, and the elimination of tissue damage and fluid shifts/losses resulting from a large trans- or retroperitoneal incision. The reduction in pulmonary complications results from performing the procedure under local anesthesia and sedation rather than general anesthesia and reducing the length of the operation.⁸ EVAR does expose the kidneys to nephrotoxic intravenous contrast, but the incidence of renal failure in EVAR patients with normal preoperative renal function does not appear to be significantly higher than in patients treated with open repair. However, special care must be taken with patients at high risk for renal complications, such as patients with preexisting chronic renal failure and diabetes.¹⁶

MIGRATION

Migration of endovascular stent grafts from an attachment site on normal vasculature toward the aneurysm sac

Figure 1 (A) Reformatted CT angiography image demonstrated stent graft in good position 30 days after EVAR. (B) Reformatted CT angiography image demonstrates caudal migration of stent graft 1 year after EVAR compared with the earlier image.

can occur at either the proximal (caudal migration) or the distal (cranial migration) end. Early aorto-aortic tube endografts had the tendency to migrate at both ends, even becoming completely dislodged into the aneurysm sac. As a result, they have become infrequently employed for endovascular AAA repair.¹⁷ Definitions of stent-graft migrations vary between authors, but usually a device is considered to have migrated if it there has been an increase of at least 5 to 10 mm in the distance between a reference point such as the lowest renal artery (for devices with infrarenal fixation) or superior mesenteric artery (SMA) (for devices with suprarenal uncovered stents) and the first visualized portion of the device on axial reconstructed computed tomography scans (Fig. 1). Cumulative rates of migration vary among authors between 3.6 and 16.6%, but the frequency of device migration appears to increase over time.^{9,18–20} Rates of migration appear to be lower among devices that have suprarenal fixation points.²¹ Factors that may predispose to device migration include continued aneurysmal degeneration of previously healthy vasculature, inadequate fixation, inaccurate sizing of the diameter of the stent graft, and abdominal trauma.^{17,18,22}

Consequences of device migration include type I endoleak, aneurysm sac pressurization and expansion, and aneurysm rupture.²² Graft migration was identified by the EUROSTAR collaborators as a statistically significant risk factor for late aneurysm rupture.⁴ Migrations that have adequate overlap with nonaneurysmal aorta may be treated conservatively with close follow-up. Device migrations are usually treated when the overlap of the aortic neck and endograft becomes less than 10 mm or upon development of a type I endoleak or aneurysm sac expansion.^{10,22}

The mainstay of treatment of device migration at the proximal neck is endovascular extension of the stent graft with aortic cuffs.^{23,24} Additionally, placement of a

Palmaz stent across the renal arteries and into the stent graft may be useful in treating patients who also have severe angulations of the aortic neck. Some authors advocate treating proximal migrations with placement of an aorto-uniiliac stent graft within the existing stent graft in cases in which deployment of an aortic cuff will be difficult or has already failed.¹⁰ Early data on treatment of proximal migration with endovascular cuffs reveal that this method of treatment has a high rate of initial technical success, but these salvage procedures have a moderate rate of postoperative failure due to continued migration or component separation.^{25,26}

Endovascular treatment of distal migrations of aorto-uniiliac or bifurcated stent grafts is similar in nature to proximal migrations in that it most often consists of deployment of an additional limb-extension endograft. These graft extensions may be placed either percutaneously or via a small groin incision.^{10,23,24} If the iliac artery is itself diseased, there may be insufficient normal artery to place a distal extension without crossing the iliac bifurcation. The iliac extension should then be placed across the bifurcation into the external iliac artery to ensure adequate seal. The ipsilateral hypogastric artery should undergo coil embolization prior to the placement of an extension endograft across its origin.¹⁰ Patients who have cranial migration of tube endografts should undergo endovascular conversion to either a bifurcated or aorto-uniiliac graft system rather than placement of a distal aortic cuff. Distal aortic cuffs for migration of tube grafts have been shown to have an unacceptably high failure rates.²³

ENDOLEAKS

Endoleaks are defined as blood flow outside of a stent-graft lumen but within the aneurysm sac and are further classified by source of the leak. Type I endoleaks result

Figure 2 CT angiography of posterior type II endoleak (white arrow) supplied by lumbar artery.

from a separation between the endograft and the arterial wall, allowing systemic circulation into the aneurysm sac. The separation may occur at the proximal or distal end of the endograft. Type II endoleaks result from retrograde blood flow from visceral arteries such as the inferior mesenteric or lumbar arteries (Fig. 2). Type III endoleaks occur from tears in the graft fabric or disconnections between modular stent-graft components. Type IV endoleaks result from perioperative graft porosity and usually resolve with correction of coagulation parameters. Type V endoleaks, also known as “endotension,” are actually continued aneurysm expansion without identifiable active endoleak.²⁷

Endoleaks are the most frequent complication after EVAR. Estimates of postoperative endoleak rates range between 15 and 23%, with type II endoleaks being most common.^{9,17,24,27} In one large trial, the mean time to endoleak development was 14.5 ± 5.7 months.²⁴ Endoleaks are most commonly detected on postoperative surveillance CT angiography but may also be detected by

Doppler ultrasonography, magnetic resonance angiography, and conventional angiography. Although CT angiography may suggest the cause and type of endoleak, often contrast angiography is needed for definitive classification.²⁸ It is important to perform selective angiography of the SMA (Fig. 3) and internal iliac arteries in addition to aortography at the proximal, midgraft, and distal positions as not all type II and distal type I endoleaks will be detected by aortography.²⁷

Endoleaks have been used as an indication of failure of treatment; however, it has been shown that endoleak status correlates poorly with changes in aneurysm volume and geometry. Although freedom from endoleak should not be interpreted as indicative of treatment success, certain endoleaks should prompt increased concern because of the resultant increased risk of aneurysm rupture. Type I and type III endoleaks expose the aneurysm sac to systemic pressurizations and have been identified by the EUROSTAR collaborators as statistically significant risk factors for late aneurysm rupture.⁴ Because of these findings, it is recommended that all type I and type III endoleaks be treated immediately after they are detected. The significance of type II endoleaks is more controversial though work has shown that the sac pressures of type II endoleak patients demonstrated systemic pressures in the majority of patients, and AAA rupture secondary to type II endoleak has been reported.^{29,30} No consensus exists on optimal management of type II endoleaks, with some physicians treating most persistent type II endoleaks whereas others choose only to treat those that are symptomatic or associated with continued aneurysm sac expansion.^{29,31}

The endovascular treatment of proximal type I endoleaks is similar in many respects to the treatment of proximal stent-graft migration. Proximal type I leaks resulting from poor seal between the device and the nonaneurysmal aorta may be treated percutaneously with

Figure 3 (A) Digital subtraction angiography (DSA) injection of SMA during an endoleak angiogram. (B) Delayed image of SMA injection reveals type II endoleak filling via inferior mesenteric artery (IMA) (black arrow).

repeat balloon dilation and/or deployment of a bare stent. If this method fails or if the proximal endoleak is associated with caudal stent-graft migration, then the endoleak can be treated with insertion of a stent-graft extension.^{8,10,17,23,24,32} Other investigators have performed brachial transarterial or translumbar induction of thrombosis with coil packing or glue of type I endoleaks that have a long length/small diameter geometry or that are too close to the renal artery for safe deployment of aortic cuffs.^{24,33,34} If endovascular techniques are not feasible or fail to treat a proximal type I endoleak, the patient can be treated with an open surgical procedure such as periaortic ligature or conversion to an open AAA repair.^{9,17,32,33}

Distal type I endoleaks also are treated in a similar fashion to cranial migration of the distal end of a stent graft. The mainstay of treatment is percutaneous insertion of iliac tube endograft extension. Again, coil occlusion of the ipsilateral hypogastric artery may be necessary if the graft extension is to be inserted into the external iliac artery.¹⁰ As with proximal endoleaks, distal endoleaks may be treated with coil embolization of the aneurysm sac if the placement of iliac extensions or open repair is not feasible. In this procedure a catheter is inserted via a femoral approach into the aneurysm sac, which is then packed with thrombogenic coils.^{24,33,34} Distal type I endoleaks that cannot be treated with endovascular methods may be treated with an open iliac artery cerclage through a retroperitoneal incision. In this procedure a balloon is inflated during the banding procedure to avoid creation of an iatrogenic stenosis in the iliac artery.¹⁰

Type III endoleaks result from junctional graft disconnections, fabric tears, or disintegration. These failures of the stent-graft system expose the aneurysm sac to systemic pressures and significantly increase the chance of late aneurysm rupture.^{4,35} Several endovascular methods are used for the treatment of type III endoleaks. In the case of poor seal between modular components, type III endoleaks will often respond to percutaneous balloon molding.^{8,12,17} Discontinuity between two pieces of a modular endograft may be treated by realigning the components by passing a guide wire through each piece and deploying an intersegmental stent graft.^{9,10,23} Fabric tears within a stent graft are rare but are often sealed with deployment of a coaxial endograft within the torn, original endograft. If endovascular methods for treating type III endoleaks fail, open conversion to surgically placed aortic graft may be performed.

The natural history of type II endoleaks is still being investigated, and as described above there is controversy regarding what constitutes the ideal treatment paradigm for type II endoleaks. However, once the decision to treat a type II endoleak is made, there are several endovascular methods available. The major-

ity of type II endoleaks arise from the transmission of blood flow or pressure into the aneurysm sac through the inferior mesenteric, lumbar, or hypogastric arteries.^{28,36} One frequently cited method of type II endoleak repair is percutaneous transarterial coil embolization of the origin of the feeder artery from a femoral or brachial approach.^{10,23,24,28,37,38} Inferior mesenteric artery endoleaks treated with this method are accessed by selectively catheterizing the superior mesenteric with a 4- or 5-F catheter and advancing a long 3-F Tracker microcatheter (Fast Tracker 18, Boston Scientific, Natick, MA) into the inferior mesenteric artery (IMA) via either the marginal artery of Drummond or the arch of Riolo. Once the aneurysm sac is reached and the site of the endoleak confirmed, microcoils are then deployed at the ostium of the IMA until the vessel is occluded.^{10,28,37,38} Lumbar endoleaks are treated in an analogous manner, with advancement of a microcatheter from the hypogastric artery through the ilio-lumbar arteries to reach the lumbar artery and aneurysm sac. Contralateral lumbar arteries are accessed through the aneurysm sac if necessary for endoleak occlusion. Coils are then used to occlude the aneurysm sac or lumbar artery ostium.^{10,23,24,38}

Type II endoleaks may also be treated by direct puncture of the aneurysm sac via a translumbar approach.^{10,37,39,40} In this approach the patient is placed in a prone position, the location of the aneurysm sac is identified by fluoroscopy and correlation of anatomic landmarks with previous CT imaging, and a route to access the aneurysm through the retroperitoneum is determined. A 19-gauge 20 cm needle is inserted into the endoleak posteriorly, several centimeters lateral to the midline at the level of the endoleak (Fig. 4). Entrance into the endoleak sac will be signaled by the pulsatile return of blood. An angiogram is then performed to confirm entrance into the endoleak. Embolization is performed with placement of coils and/or liquid adhesive such as N-butyl cyanoacrylate "glue" into the endoleak sac.^{10,37,39} Depending on the anatomy of the IMA and lumbar arteries, there are some cases where feeding vessels can be selected and embolized via the translumbar approach. Translumbar endoleak embolization is preferably performed via a left translumbar approach but can be safely performed using a right (trans-inferior vena cava) approach when needed.⁴¹

One cohort study found an 80% (16/20 patients) failure rate of IMA endoleaks treated with transarterial coiling compared with an 8% (1/13 patients) failure rate for patients treated with translumbar embolization.³⁷ The authors hypothesized that endoleaks may behave in a manner analogous to arterial malformations, and therefore, occlusion of one feeding artery by methods such as transarterial endoleak embolization fails to treat the lesion because of subsequent recruitment of additional collateral feeders.

Figure 4 (A) DSA of type II endoleak obtained via translumbar catheter (black arrow). (B) This endoleak has been treated via a translumbar approach using coils.

Transarterial embolization can be accomplished not only by selecting the feeding vessel but also by entering the endoleak itself. This technique allows for transarterial coil embolization of the aneurysm sac in a manner similar to the translumbar approach. The catheter can then be withdrawn and used to embolize the feeding vessel. This technique allows for embolization of the aneurysm sac as well as the feeding vessel. Patients who fail endovascular treatment of a type II endoleak may undergo either laparoscopic clipping of the inferior mesenteric and lumbar arteries or conversion to a surgically placed graft with suture ligation of these vessels.^{4,42}

GRAFT LIMB COMPLICATIONS

Graft limb complications include kinking, stenosis, or thrombosis of the stent-graft limbs as well as fabric twisting and redundancy in devices with unsupported limbs. One large multidevice case series reported graft limb complications in 2.8% of patients postoperatively, and these failures were predominantly early in the follow-up period with no failures after 30 months.⁹ Another case series with the unsupported Ancure graft (Guidant Corporation, Indianapolis, IN) reported an 11% reintervention rate for limb failure, and 82% of the postoperative limb complications occurred within the first 6 months of device implantation.⁴³ Graft limb occlusions can develop due to device compression in a long, narrow proximal aortic neck, at a calcified aortic bifurcation, in tortuous iliac arteries, or secondary to poor distal runoff in patients with comorbid peripheral vascular disease. Alteration in aneurysm geometry with sac shrinkage also contributes to late kinking or stenosis of stent grafts, and in these cases, supported stent grafts are more likely to develop occlusions because they are less compliant than unsupported devices.^{12,17,43} Graft limb occlusions and thromboses are more likely to be detected clinically when the patient presents with acute onset of pain, paresthesias, paralysis, and/or absence of a

femoral pulse rather than by radiological means, but stenoses may be detected on follow-up CT.⁴⁴ Treatment of graft limb complications is indicated to avoid ischemic sequelae resulting from limb stenosis, thrombosis, or occlusion.

The majority of graft limb complications are treated percutaneously with angioplasty, bare stent placement, or thrombolysis. Fabric twisting in unsupported stent grafts may be treated with self-expanding stent deployment to relieve any stenosis and to prevent further twisting of graft material.¹⁰ Kinking in supported or unsupported devices also is treated with balloon angioplasty and stent placement.^{10,12} When treating a lesion in one limb of a bifurcated graft that lies proximal to the native aortic bifurcation, deployment of a “kissing stent” in the contralateral limb may be necessary to avoid compression of the unstented graft limb.^{44,45} In cases of severe graft kinking in which stent deployment is not feasible or has failed, conversion to an aorto-uniiliac graft and femoral-femoral extra-anatomic bypass with placement of an occluder device within the kinked limb may be indicated.¹⁰ Stenoses within stent grafts may occur for reasons beyond kinking, such as vessel tortuosity and occlusive disease. Stenoses resulting from either of these etiologies are treated in an identical manner to stenosis due to kinking, with balloon angioplasty and stent deployment.⁴⁴

Thrombosis of stent-graft limbs can be treated with percutaneous intra-arterial thrombolysis. If the lysis procedure is successful, the underlying lesion, usually kinking or stenosis, or any downstream occlusive disease should be treated with balloon angioplasty and stent placement.^{10,44–46} Graft occlusion with thrombus may also be treated mechanically with a surgical thrombectomy through a femoral arteriotomy. It has been suggested that this treatment method should be used with caution, as the balloon thrombectomy may cause disruption of the endograft.⁴⁴ Another author reported only a 50% success rate (two of four patients) for graft limb

Figure 5 (A) DSA of patient with acute oliguria after EVAR secondary to covering the right renal artery with the covered portion of the suprarenal stent graft. (B) The right renal artery has been revascularized by extending the renal artery stent into the uncovered portion of the stent graft.

thrombosis treated with chemical thrombolysis, and therefore advocates femoral-femoral bypass as the treatment of choice for patients with a single thrombosed graft limb.¹⁰

RENAL ARTERY OCCLUSION

Renal artery occlusion following EVAR can occur by several mechanisms. The most common cause of renal artery occlusion is perioperative deployment of the aortic stent graft so that it covers either one or both renal arteries. Patients who have aneurysms with short necks may be at especially high risk for this complication.⁴⁷ The rate of renal artery occlusion has been reported to be between 2 and 4% upon initial deployment of the stent graft.^{7,48} Renal artery occlusion may also develop after intraoperative rupture and embolization of cholesterol plaques, which occurred in 3.3% (3 of 90 patients) in one series. A further mechanism of renal artery occlusion is occlusion after aneurysm remodeling.⁷ Patients who develop renal artery occlusion during or after EVAR can undergo treatment to reopen the artery. Failure to quickly reestablish circulation to the kidney can lead to ischemic damage and infarction as the kidney has a limited warm ischemic time.⁷

Treatment of renal artery occlusion due to graft maldeployment via a “pull-down” method has been described. In this method a stiff guide wire is inserted through a femoral sheath, advanced over the graft bifurcation, and externalized through the contralateral femoral sheath. By applying force in the caudal direction to both ends of the catheter, the stent graft can be moved distally until it no longer covers the renal artery orifice. This technique can be successfully employed even in

devices that have proximal fixation hooks.^{47,49,50} Plaque embolization causing renal artery stenosis may be treated by catheterization of the renal artery and aspiration of the plaque.¹⁶ A renal artery that is covered by a stent graft either immediately after placement or due to chronic alteration of aneurysm geometry may be treated by insertion of a bare stent into the renal artery⁹ (Fig. 5).

CONCLUSION

The perioperative (and perhaps long-term) advantage of EVAR is based upon sparing the patient from an open surgical procedure and in some cases general anesthesia. The trade-off for the less physiologically stressful repair is that EVAR appears more prone to graft-related complications. Many of the complications unique to EVAR can be repaired using endovascular techniques. The next step that is needed is further research investigating the long-term results of endovascular treatments of stent-graft complications such as endoleak embolization, endograft extensions, and graft limb angioplasty and stenting.

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