OPINION

Transluminal repair of abdominal aortic aneurysm: A call for selective use, careful surveillance, new device design, and systematic study of transrenal fixation

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A growing worldwide experience with transluminal endografting (TE) of abdominal aortic aneurysms (AAAs) and the United States Food and Drug Administration's late 1999 approval of two devices for commercialization have combined to produce a surge of interest in this less invasive form of therapy.[1-24] The brief training programs provided by the manufacturers have served to increase the number of operators and clinical sites involved. But the proper place of TE in the armamentarium of AAA management has yet to be elucidated, and a variety of major problems and limitations of the new method have begun to surface. In addressing these problems, it should be possible from the available published data to formulate plans for clinical investigation and technology development that will advance knowledge and practice in TE through attempts to overcome existing shortcomings. In the following opinion, patient selection, posttreatment surveillance, current design limitations, and a rationale for careful study of transrenal fixation are all addressed.

The late survival rate after successful open surgical AAA repair has been reported to be approximately 67% at 5 years.[25] Coronary heart disease contributes significantly to both perioperative morbidity and mortality rates and to late mortality rates in patients who undergo treatment.[26-33] Pulmonary disease, renal dysfunction, and advanced age also add risk. For conventional AAA repair, the published results document a 3% to 7% perioperative mortality rate among unstratified patients who undergo treatment with elective surgical repair[34-36] and the following risk-stratified perioperative mortality rates: 0 to 1% among patients in The Society for Vascular Surgery and The International Society for Cardiovascular Surgery risk stratum 0, 1% to 3% among patients in risk stratum 1, 3% to 8% among patients in risk stratum 2, and 8% to 30% among patients in risk stratum 3.[37,38] In addition, results of a recently published report of 16,450 patients (average age, 72 ± 7 years) from the Nationwide Inpatient Sample who underwent elective open surgical AAA repair during the period from 1994 to 1996 revealed an in-hospital mortality rate (underestimation of the true total perioperative or 30-day mortality rate) of 4.2% and an overall complication rate of 32.4%.[39] The perioperative mortality rate increased with age, with female gender, with cerebrovascular occlusive disease, with preoperative renal insufficiency, and with more than three comorbidities. It is easy to see why, after each preoperative evaluation, a skilled clinician and informed patient together must decide whether and when to operate, with the weight of evidence from the literature and the patient's individual risk assessment as guides. Obviously, not all patients have ideal or even suitable conditions for open AAA repair. What, then, becomes of the patients who do not undergo treatment?

Recently, Conway et al[40] reported a 10-year follow-up study in which 106 patients with AAAs of more than 5.5 cm in diameter did not undergo treatment. At the end of the study, 76 patients (71.7%) had died.[40] In this select study population of patients with conditions that were unsuitable for surgery, the poor 10-year survival rate is not exactly a revelation. However, the study results also revealed AAA rupture as the cause of death in 36% of the patients with AAA diameters of 5.5 to 5.9 cm at baseline, in 50% of the patients with AAA diameters of 6.0 to 7.0 cm at baseline, and in 55% of the patients with AAA diameters of more than 7.0 cm at baseline. The median survival rate in the latter group was 9 months. How might the specific management chosen and the long-term survival rate

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in this study have been altered by the availability and application of TE?

Two United States Food and Drug Administration–approved AAA endografts and several other investigational ones are in use in the United States today. Because TE is relatively new and because comparatively few studies have included mid-term and late outcomes,[4,5,7,10,15,17,22-24,41] significantly less is known about TE than about conventional operation.[39,42-47] Important questions remain about the durability and completeness of protection from AAA rupture provided,[22,41,48,49] about late complications as the result of changes in AAA morphology[23,24,50-54] or as the result of endograft failure, about intermediate-term and long-term patency rates,[55] and about the role of TE in subgroups that range from the highest risk with shortest infrarenal aortic necks[56,57] and other suboptimal anatomy to the lowest risk with younger healthier patients with small AAAs.[58,59]

Although TE is appealing because it is inherently less invasive than conventional open repair, it has its own shortcomings, one of the most important of which is the problem of endoleaks.[22,41,48,49,53,60,61] Although they are not unique to TE, late post-treatment AAA ruptures do occur and have become a cause for concern.[61-64] From the results of the limited reports available, it appears that late AAA rupture after TE occurs in 1% or fewer patients. However, long-term follow-up results comparable with those of open repair are still not available. Therefore, this number may be falsely low. Nevertheless, it is already clear that although TE confers durable protection against AAA rupture on treated populations, that protection is not absolute in patients with endoleaks because late AAA enlargement and even rupture can occur in these patients. Given current knowledge, technology, and practice, close surveillance after TE with the inclusion of contrast-enhanced spiral computed tomographic (CT) scanning is essential and has been emphasized by the United States Food and Drug Administration.[63]

Late ruptures after apparently successful TE are complex phenomena with potentially multifactorial causes. The following factors are among the potential contributions to the problem in any given case: 1, poor patient selection; 2, operator/procedure-related factors; 3, device-related factors; and 4, anatomic-pathologic factors. Whatever the causes of late AAA rupture after TE, the facts and theory to date invoke endoleakage as the common underlying pathophysiologic state that allows the AAA sac to remain pressurized (early or immediate endoleak) or to resume a pressurized state (late-onset endoleak). Type IA (proximal attachment) endoleaks are considered to be the most ominous.[60] Endoleakage shown with imaging is not universally accepted as a precondition for AAA enlargement after TE because enlargement and rupture have been known to occur in patients whose serial CT scan results failed to show endoleaks. Increased pressure has been documented in AAA sacs after TE in the absence of endoleaks shown on CT scan results.[50] Such experiences have led to the use of the term, "endotension," which refers to the situation of a pressurized AAA sac after TE without endoleak on CT scan results.[41] Experts in CT scan imaging, however, have criticized reports of CT scan results that fail to document endoleaks in patients with growing AAAs. They emphasize the importance of specific CT scan imaging protocols that are adequate for the detection of endoleaks.[65]

Poor patient selection involves inclusion of the following patients: patients with no infrarenal aortic neck or pyramidal shape of aortic neck from renal arteries caudad to the level of the AAA; those patients with extensive thrombus or plaque in the infrarenal aortic neck, which often indicates that the "neck" is involved with the AAA and that there is no adequate attachment zone; and those patients with marked angulation between the infrarenal aortic neck and the main axis of the AAA or between the upper abdominal aorta and the infrarenal neck. In the process of deployment, the latter condition may result in the proximal endograft attachment "seeking" but not "finding" the entire wall for anchoring. The result may be caudal displacement of the proximal attachment and a potentially compromised proximal seal. Depending on the particular design, marked angulation may also compromise endograft function in other ways (see subsequently).

The operator/procedure-related factors that may be responsible include the following conditions: inadequate oversizing of the proximal attachment for the diameter of the infrarenal aortic neck, excessive oversizing of the proximal attachment (may cause pleating of the graft and resultant type IA endoleak), lower-than-ideal placement of the proximal attachment in the infrarenal aortic neck, improper sizing of the distal ends of the graft endolegs in ectatic/aneurysmal iliac vessels (type IB endoleak), insufficient overlap of modular graft components (type III endoleak), and other deployment errors.

The device-related factors include the following conditions: modularity instead of unibody construction; lack of an attachment mechanism for the prevention of migration; potential aortic wall weakening as the result of continuous pressure from expanded attachment devices, which may lead to an increase in infrarenal aortic neck diameter and late type IA endoleak; changes in graft position or in the relative positions of components over time because of either multiple cycles or actual morphologic change in the AAA and aorta after initially successful exclusion; other changes in position that result from kink resistance or column strength in the presence of changing AAA and aortic morphology; graft fabric holes or tears as the result of failure of the graft material or damage to the graft material by intact or fractured (caused by metal fatigue) metallic members of the device or even calcified plaque contacting the graft material; failure of attachment components; and finally obligatory infrarenal attachment (no transrenal/suprarenal attachment option) because of endograft design. In our own experience,[61] type IA endoleaks have occurred in 3.6% of the cases that involve transrenal fixation versus in 5.0% of the cases that involve infrarenal fixation, for an odds ratio of 1.4.

The anatomic-pathologic factors that predispose a patient to endoleak, AAA enlargement, and potential rupture include the following conditions: lack of complete incorporation of the attachment components into the aortic wall;[66] large collateral anastomoses[65] between the iliolumbar branches of the internal iliac artery and the lumbar branches of the aorta, which lead to type II endoleak; native arterial anastomoses between the left branch of the middle colic artery and the left colic branch of the inferior mesenteric artery, with a patent inferior mesenteric trunk arising from the aorta, which also lead to type II endoleak; shortening of the AAA or the aorta and iliac arteries after TE, with resultant migration or relative migration of the endoprosthesis or its components, which may cause type IA, IB, or III endoleak; and finally continued enlargement in the diameter of the AAA infrarenal neck after treatment that is attributable to aging or to the disease itself. Obviously, the latter condition can result in late type IA endoleak, AAA enlargement, and rupture. Other investigators have established that enlargement of the AAA infrarenal neck after treatment without enlargement of the suprarenal aorta can and does occur[51,67] and may be associated with endograft migration and late endoleak.[23,68] Infrarenal aortic neck dilatation occurs even after conventional operation.[69] In one small CT scan study of 19 patients who underwent open AAA repair, Sonesson and colleagues[70] showed that the infrarenal neck, the aortic diameter at the level of the renal arteries, and the diameter at the level of the superior mesenteric artery all had increased at a mean follow-up period of 6 years after surgery but that the rate of increase was the least above the renal arteries. AAA neck enlargement may be limited or pose no danger in the presence of a suture anastomosis of conventional open repair. However, the potential danger is obvious in the case of TE. Unfortunately, it is unclear how to reliably predict which infrarenal aortic necks will enlarge the most or the fastest after endografting and which patients will have type IA endoleaks develop. Not surprisingly, thus far, we are also unable to answer complex questions, such as whether increased AAA sac pressures associated with type II endoleaks[50] can, with time, contribute to infrarenal neck dilatation and the onset of late type IA or type III endoleaks. All of these facts and questions underscore today's dependence of operators and patients on the specific attachment mechanism. However, because the usual situation in practice today finds the operator lacking control over endograft features and their mechanisms of operation, the specific attachment site that is chosen looms as extremely important.

With all of the aforementioned, there is an abundance of rationale for further study of transrenal endograft fixation. Endografting immediately below the renal arteries, with use of bare metal spring attachments across the renal arteries and onto the suprarenal segment of the aorta, offers the greatest potential (given currently available technology) to minimize concerns about late enlargement of the infrarenal aortic neck. Branched endograft designs are forthcoming,[71] but for now they cannot be considered an option in TE of AAAs.

Little has been written about the results and consequences of transrenal fixation.[56,57,72,73] Malina and colleagues[72] crossed 25 renal arteries with bare metal spring in 18 patients with AAAs who underwent endografting. In their follow-up period to a median of 6 months, all the arteries remained patent, there was no effect on serum creatinine levels, and only one patient had evidence of a small infarct in one kidney on follow-up CT scan results. In a series by Bove and colleagues[73] with a median follow-up period of 6 months, 28 patients underwent AAA treatment with the use of the Talent LPS bifurcated endograft (Medtronic, Sunrise, Fla) with use of transrenal fixation. No renal infarcts were detected with follow-up CT scanning, and only one patient of the 19 with healthy preoperative renal function had a persistent elevation of creatinine level after surgery.

In our own experience, 222 patients (73%) underwent endgrafting with infrarenal fixation (IF) and 83 (27%) underwent transrenal fixation (TF). Preoperative and postoperative serum creatinine levels and follow-up serum creatinine levels were obtained. These laboratory values and inspection of the renal parenchyma on follow-up AAA CT scans in a subset of 100 patients failed to yield convincing evidence of embolic problems or of a negative impact of TF on renal function. Overall, creatinine clearance declined an average of 5 mL/min in the transrenal group but remained stable in the infrarenal group. Nevertheless, the sample size was small, patient characteristics differed between the infrarenal fixation and TF groups, and the data were not definitive. Clearly, more studies of TF are needed.

In summary, several related phenomena late after TE, including infrarenal aortic neck enlargement, type IA endoleaks, AAA enlargement, and AAA rupture, firmly establish the rationale for seeking the renal and suprarenal segments of the aorta as the ideal sites for proximal fixation. However, it is unclear whether TF negatively impacts renal function and, if it does, whether the impact outweighs the apparent advantage of TF. Thus, studies are urgently needed to track and describe in detail the changes that occur in the aorta and in the kidneys after endovascular AAA repair with TF, with the Talent LPS, the Cook Zenith endograft (Cook, Inc, Bloomington, Ind), and other suitable endograft designs. Ultimately, it may prove important to include in these studies subjects from all The Society for Vascular Surgery and The International Society for Cardiovascular Surgery risk strata 0 through 3. However, given currently available technology and results, the youngest patients with AAA with the greatest life expectancy still remain the best candidates for open AAA repair and the poorest candidates for TE. Most importantly, there is much more needed than just additional clinical data. There is a glaring clinical need for technologic improvements in the proximal fixation mechanisms of endografts. In the future, perhaps newer endograft designs with excellent attachment mechanisms and low frequencies of late failure will provide better therapeutic options to most patients with AAA.

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