Comparing Endovascular and Open Repair of Abdominal Aortic Aneurysm

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To the Editor: The report of the short-term outcomes of the Open Versus Endovascular Repair (OVER) trial by Dr Lederle and colleagues provided little information regarding the endovascular procedures used for repair of abdominal aortic aneurysm (AAA). Although the open surgical procedure may have become standardized during the last 3 decades, with homogenous results regardless of the manufactured graft type, the same is not likely with the endovascular stent grafts.

The different types of endografts have diverse anatomical and morphological features leading to differing technical feasibility, varying types of construction, and differing materials. Therefore, they are made to treat different types of patients. The authors stated that all patients were eligible for both open surgical and endovascular procedures, but did not describe the specific endovascular inclusion criteria and the anatomical characteristics of the patients who were treated.

Moreover, the authors reported endoleaks in 25% of the endovascular group, resulting in a secondary procedure in 4.1% of patients. However, they did not give information regarding the actual types of endoleaks (which can vary from the “innocent” type II endoleaks to “aggressive” type I and type III endoleaks), the percentages of endoleaks by the different types of endografts, or the fate of the leak and the aneurysm sac in the remaining 23.9% of the patients under observation.

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To the Editor: Dr Lederle and colleagues conducted a randomized trial to compare endovascular vs open repair of AAA, showing lower perioperative mortality for endovascular repair. The early advantage of endovascular repair was not offset by increased mortality in the first 2 years after repair.

The atherosclerotic process is often not limited to a single arterial location, giving it a character of a systemic and generalized disease. More than 25 years ago, a study by Hertzer et al demonstrated that only 6% of patients with an AAA have a healthy coronary tree. In a study by Feringa et al, a group of patients who had vascular surgery and underwent preoperative cardiac testing had an asymptomatic ejection fraction of less than 40% or silent ischemia (new wall motion abnormalities) in 14% and 41% of the patients, respectively.

Although the atherosclerotic process may generally remain asymptomatic, surgical stress may elicit a rapid progression of the atherosclerotic disease. This progression is reflected by asymptomatic perioperative troponin T release, an important marker of underlying coronary artery disease. Studies have demonstrated prevalence of troponin T release of 10% after endovascular repair and 30% after open repair, with up to 90% of the troponin T elevations asymptomatic. The occurrence of asymptomatic perioperative myocardial damage, assessed with troponin T measurements and continuous electrocardiographic monitoring for 72 hours, has been associated with a 2.3-fold increased risk for long-term mortality in patients who have had vascular surgery.

Endovascular repair of AAA may result in a reduced perioperative stress response compared with open repair, which could explain the reduced short-term mortality rates. The disappearance of the early advantage of endovascular repair after 2 years could be explained by a high incidence of asymptomatic coronary artery disease, with an accelerated subclinical progression due to surgical stress that results in asymptomatic perioperative cardiac damage and reduced survival rates during long-term follow-up.

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To the Editor: In their article reporting the short-term perioperative outcomes of the OVER trial, Dr Lederle and colleagues1 inaccurately stated, “All 109 lead proceduralists for aneurysm repair were vascular surgeons.” They also commented, “Procedures in our trial were performed by experienced university-affiliated vascular surgeons,” suggesting this as an explanation for improved perioperative outcomes in the OVER trial relative to the EVAR-1 and DREAM trials. These statements overlooked and obscured the contribution of many vascular interventional radiologists, some of whom were investigators in the trial.

In my own experience, participating in the care of these patients in 2 of the centers (George E. Wahlen VA Medical Center, Salt Lake City, Utah, and Puget Sound VA Medical Center, Seattle, Washington), the lead proceduralists, providing oversight and direction and personally performing the majority of the endovascular aneurysm repair procedures, were vascular interventional radiologists. In 2002, when randomization began in the OVER study, very few vascular surgeons had the training and experience to independently perform endovascular aneurysm repairs, most of which were being performed by vascular interventional radiologists. During the period of enrollment, between 2002 and 2007, vascular surgery fellowships increasingly included formal training in catheter intervention, including endovascular aneurysm repair. Therefore, toward the end of the enrollment period, vascular surgeons were more likely to perform these procedures independently.

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In Reply: We agree with Dr Dalainas that the different endovascular systems are made to treat somewhat different patients. This is why at randomization we recorded the system that would be used if patients were assigned to endovascular repair to ensure comparison with patients assigned to the appropriate open repair in the subgroup analysis. The inclusion criteria were as stated in the article, with the pertinent issues being the AAA diameter and the requirement that patients had to be considered a candidate for both procedures by the participating vascular surgeon and meet the manufacturer’s indications for the endovascular system that would be used if so assigned. Although the manufacturer’s indications are quite specific, the study inclusion criteria could not be because they had to accommodate endovascular systems that were not yet approved at the time the study was planned. We also intended that inclusion be as unrestricted and reflective of usual practice as possible. In response to Dalainas’ request for more detail on endoleaks, our article focused on the comparison of clinical outcomes after open and endovascular repair, such as secondary procedures, some of which resulted from endoleaks. However, endoleaks per se are not a clinical outcome that can be meaningfully compared with open repair outcomes. Detailed descriptions of endoleaks and anatomic characteristics of enrolled patients may be included in future analyses.

In their discussion of “the disappearance of the early advantage of endovascular repair after 2 years,” we are uncertain whether Dr van Kuijk and colleagues are referring to the earlier European trials, in which the survival curves converged, or to our study, in which the loss of statistical significance at 2 years resulted not from excess late deaths after endovascular repair but from the decrease in the relative difference in mortality rates as the total number of deaths increased, a phenomenon requiring no further biological explanation. We do not agree with an assumption that AAAs are a manifestation of the atherosclerotic process, as the preponderance of data appear to suggest a distinct etiology.1

We thank Dr Findeiss for calling attention to the role of interventional radiologists in our study, and apologize to her and her colleagues for failing to adequately acknowledge their contribution. Our data on lead proceduralists were provided by the centers and indicated the person responsible for the study for the procedure, and so may not have fully captured individual contributions. We have subsequently reviewed operative reports at the 6 centers with study-approved interventional radiologists and identified 42 endovascular repairs in which interventional radiologists had a major role. We are extremely grateful for their participation, and the expertise of these interventional radiologists clearly contributed to the success of the study.

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