Identification and implications of transgraft microleaks after endovascular repair of aortic aneurysms

Jon S. Matsumura, MD,a Robert K. Ryu, MD,a and Kenneth Ouriel, MD,b Chicago, Ill, and Cleveland, Ohio

Purpose: The purpose of this report is to describe an interesting cause of endoleak and detail-specific techniques for identifying small transgraft defects, which we have termed microleaks.

Methods: Four patients underwent endovascular repair of abdominal aortic aneurysms with modular nitinol/polyester endoprostheses and were studied after 6 to 30 months. All patients were enrolled in standard follow-up radiographic surveillance protocols.

Results: Three of the four abdominal aortic aneurysms continued to expand after endograft repair. Standard computed tomography imaging with precontrast, dynamic contrast, and delayed imaging frequently identifies endoleak, although it fails to precisely identify microleaks as the source. Color flow duplex ultrasound scan was performed on three patients and perigraft “jets,” small areas of color flow adjacent to the endograft, were identified in all. Microleaks were identified in one patient who underwent digital subtraction arteriography with directed efforts to completely opacify the prosthesis lumen and multiple oblique projections. In another patient, contrast arteriography with balloon occlusion of the distal endograft clearly depicted midgraft microleaks that might otherwise be mistaken for graft porosity or cuff junction endoleaks. No microleaks were diagnosed on angiograms when these directed efforts were not performed. Aneurysm exploration before aortic clamping provided conclusive determination of the presence of blood flow through the wall of the endoprosthesis in two patients.

Conclusions: Microleaks occur up to 2.5 years after endovascular repair of aortic aneurysms. Although computed tomography demonstrates the presence of an endoleak in these patients, the exact site of origin usually remains obscure. Doppler ultrasound scan and directed arteriography appear to be of greater utility for identifying the presence and location of microleaks. Balloon occlusion arteriography and aneurysm exploration without arterial clamping provide definitive evidence of microleaks. Although the clinical significance of microleaks remains unclear, long-term monitoring of patients is imperative to diagnose and treat these and other modes of endograft failure before they progress to aneurysm rupture. (J Vasc Surg 2001;34:190-7.)

Endovascular repair of abdominal aortic aneurysms (AAA) has stimulated considerable interest since initial reports were published almost a decade ago. There are clear short-term benefits compared with standard open repair in terms of reduced blood loss, fewer complications, and shorter hospital stays.1-3 However, there is almost uni-
cal trial under an investigational device exemption, and one patient was treated with the system after its marketing had been approved by the US Food and Drug Administration. All patients were studied with Institutional Review Board approval. All were followed up clinically and radiographically with a postoperative surveillance protocol. This protocol involved periodically taking each patient’s history and determining results of a physical examination, abdominal radiographs, and contrast-enhanced helical computed tomography (CT) scans with precontrast, dynamic contrast, and delayed-contrast imaging. Follow-up protocols varied over time, particularly with regard to the frequency of CT scans in patients with endoleaks. Duplex ultrasound

**Fig 1.** Color flow duplex scan of patient A. Doppler assessment of small jets of flow that could be easily misinterpreted as artifact or branch endoleaks.

**Fig 2.** Late phase of arteriogram of patient A showing contrast pooling in aneurysm sac.

**Fig 3.** No distinct source of endoleak is identified from graft with standard digital subtraction angiogram with power injector and pigtail catheter.
scan was substituted or performed in addition to CT scans in selected patients. Patients were taking aspirin, and one was taking anticoagulants.

Patient A is a 74-year-old man who underwent repair in October 1997 for a 5.5-cm AAA, with the main trunk of the prosthesis placed from the left femoral approach. Initial CT before discharge showed contrast inside the aneurysm and outside the graft consistent with transgraft porosity. Over the next 12 months several CT studies were performed and interpreted locally to show endoleaks separate from the attachment sites, probably related to lumbar arteries, that were inconsistently identified near the graft bifurcation and left (ipsilateral) iliac limb. At 7 months, an increased cross-sectional area of contrast enhancement was seen outside the graft. Duplex scanning had revealed small areas of color flow adjacent to the graft (Fig 1) that were interpreted as branch endoleaks. At 1 year, arteriography revealed an endoleak from the left limb of the graft as well as lumbar endoleak, and an additional iliac extender cuff was placed within the previous ipsilateral left limb. Subsequent CT scans were interpreted locally as demonstrating stable AAA size but continued endoleak, although the appearance of the perigraft contrast continued to change and became smaller at the 24-month time point.

Investigators at the core laboratory reviewed several CT scans and initially reported the leak as originating from the graft junction and collateral branch flow, with subsequent reports of only collateral branch flow and no endoleak. Investigators at the core laboratory reported a 7-mm enlargement of the AAA between the 12- and 15-month time point with indeterminate source endoleak, which coincided with the period after placement of the second endograft. Frequently, there was discrepancy between core laboratory and local interpretation, the latter of which formed the basis for most clinical decisions.

At the 30-month visit, a 4-mm AAA enlargement was noted, and repeat digital subtraction arteriography demonstrated foci of contrast accumulation in the AAA sac at several levels (Fig 2), although no clear hole was identified (Fig 3). Selective hypogastric arteriograms revealed lumbar artery endoleaks, although the collateral pathway included very small vessels, and operative exploration was elected.

At the time of laparotomy, there was minimal pulsation in the AAA. Pressure measurements by direct puncture of the sac with a needle, which was intentionally directed by the surgeon lateral to the endograft, showed mean blood pressure equal to the radial artery, but a dampened pulse pressure. Vascular clamps were placed near the proximal and distal necks, and the left iliac artery was clamped before the AAA was opened. Exploration of the sac revealed pulsatile bleeding through a small hole in the right (contralateral) limb of the endoprosthesis before the aorta and other iliac artery were cross-clamped (Fig 4). There was loose, partially liquefied thrombus in this region. The proximal and distal ends were well incorporated in the native arteries, and an endarterectomy of the infrarenal aorta and both common iliac arteries had to be performed to remove the entire prosthesis. In contrast, the gate junction separated easily during explantation. Lumbar artery back bleeding was less than from the graft although it was observed only after the cross-clamps were placed. A standard bifur-
cated endoaneurysmal repair was performed, and the patient recovered uneventfully. Examination of the explanted prosthesis revealed several areas where the sutures that attached the fabric to the exoskeleton were broken or missing (Fig 5); these were concentrated at the regions where the aneurysm sac joins the infrarenal neck and iliac arteries. The explant was prepared and returned to the manufacturer according to the study protocol.

Patient B is a 73-year-old man who underwent endovascular repair in November 1999 for a 6.2-cm AAA. The initial CT scan showed no endoleak and an air bubble in the aneurysm sac. His 6-month CT scan showed an endoleak near the midportion of the right limb (Fig 6), a large endoleak near the native aortic bifurcation (consistent with a distal endoleak), and movement of the graft within the AAA. The AAA size did not change significantly in this interval. Arteriography revealed a Type I endoleak from the distal left iliac attachment site. Selective right iliac limb arteriography with 3-second powered contrast injections at 8 mL/s in multiple oblique magnified views demonstrated a microleak from the midsection of the right iliac limb (Fig 7). The patient was treated on the left with a “bell-bottom technique”; an aortic extender reinforced by a subsequent iliac limb to sandwich the short overlap of the aortic cuff was used. On the right side, a coaxial iliac endograft was placed within the previously ipsilateral limb. Although smaller, there was persistent indeterminate endoleak on the predischarge CT. If there is continued enlargement in follow-up, then further directed studies and reintervention will be considered.

Patient C is an 83-year-old man who underwent repair in May 1998 for a 5.2-cm AAA. He had no symptoms, no pulsatile mass was felt during examination, and follow-up CT showed the AAA size was stable. At 24 months, the AAA was noted to be pulsatile again, and CT showed enlargement of the AAA to 5.6 cm. The contralateral limb was initially placed correctly within the box formed by the four radiopaque gate markers, and this was documented on the first postoperative radiograph. There was subsequent

---

**Fig 5.** Photograph of graft, after explantation with its accompanying trauma, and rinsing. Fabric material is separated from stent frame, and broken sutures (black straight arrows) are visible. There is a striking difference of tissue incorporation in midgraft compared with end implanted in iliac artery on left side of picture.

**Fig 6.** CT scan showing endoleak (black arrow) intimately associated with middle segment of right iliac limb.

**Fig 7.** Oblique magnified digital subtraction angiogram demonstrating microleak (black arrow) from midgraft.
caudal migration of the iliac limb relative to the gate junction seen on abdominal films. Although there was no dislocation in this case, gate migration could result in a classic Type III endoleak, and initial placement as cranial as possible in the gate is now performed to allow the longest segment of overlap. A duplex ultrasound examination revealed microleaks of both iliac limbs (Fig 8).

In the operating room, arteriography was performed with balloon occlusion of flow in the right iliac limb, permitting manual injection of full-strength radiocontrast media through the guidewire lumen. This revealed contrast flowing out of the end hole, caudal down the occluded iliac limb, out a midgraft microleak (clearly apart from the gate junction), and into the AAA sac without outflow (Fig 9). Subsequent arteriography with distal occlusion of the left iliac limb did not reveal a microleak although residual contrast within the sac may have obscured visualization. Coaxial iliac limb endoprostheses were placed bilaterally to overlap the separating gate junction and to seal the bilateral microleaks. The first follow-up CT showed dense residual contrast in the sac, outside the endograft on preinfusion imaging.

Patient D was a 71-year-old man who underwent repair of a 7.0-cm AAA in November 1998. The body and ipsilateral limb were placed through a left femoral approach. An iliac extension limb was placed to seal the right iliac attachment site. A CT scan performed on the first postoperative day revealed a presumed Type II endoleak thought to be originating from a patent inferior mesenteric artery (IMA). The patient was followed up without further intervention and was receiving long-term oral anticoagulation. In December 1999 a CT scan documented growth of the AAA to a diameter of 8.0 cm. Arteriography was performed, with selective injections of each iliac limb. Only a single small puff of contrast through the left iliac limb was detected. This area was in immediate juxtaposition to the end hole of the catheter, and the injection itself was thought to account for the finding. A selective cannulation of the superior mesenteric artery was performed; injection demonstrated retrograde flow of contrast through the IMA into the aneurysm sac. The IMA orifice was coil occluded through the superior mesenteric artery access site, but the endoleak persisted on CT. A subsequent CT was performed in May 2000 and demonstrated growth of the AAA to 9.0 cm in diameter. Less than a month thereafter, the patient presented with acute onset of severe, unrelenting back pain, and vascular surgery was consulted. An urgently performed CT scan demonstrated a new collection of contrast material in apposition to the right iliac limb and inflammation or extravasated blood overlying the anterior surface of the aneurysm. The patient was taken to the operating room where, through a left retroperitoneal approach, an unruptured aneurysm encased in an inflammatory peel was exposed. The aneurysm was tense, but nonpulsatile. After control was gained at the suprarenal level, the aneurysm sac was opened before arterial clamping. Two pulsatile jets of blood flow were observed: one was originating from the midpoint of the right iliac limb (Fig 10) and a second, smaller jet from the left iliac limb. The proximal and distal attachment sites were dry, and there was no back bleeding from lumbar arteries or the IMA. The entire device was explanted, and the aneurysm was repaired with a conventional polyester prosthesis. Initially, the patient did well,
but had respiratory insufficiency requiring reintubation on the third postoperative day. On the sixth postoperative day the family requested withdrawal of respiratory support, and the patient died. Gross pathologic evaluation of the graft failed to reveal large defects in the graft material, although several sutures were broken and tiny defects were found at the site of the suture holes.

DISCUSSION

This report provides preliminary findings of persistent focal transgraft endoleaks that have not been previously reported. These microleaks are noteworthy in that they are present up to 2½ years later and may persist despite the absence of anticoagulation. Of greatest concern, however, is the association of microleaks with continued growth of the aneurysm on later follow-up. Aneurysms that enlarge after endovascular repair deserve careful study with sensitive diagnostic tests, and strong consideration should be given to conversion if no satisfactory endovascular treatment can be accomplished.

It is puzzling that these microleaks have so far escaped detection despite multicenter studies with core laboratory review and thousands of implants worldwide. One possible explanation is that they are small and have been identified but discounted as graft porosity by investigators and physicians.12 Microleaks may be misinterpreted as Type II endoleaks, which may have occurred in patient D. Furthermore, core laboratories may identify many microleaks, but the availability of their report is often delayed and clinicians may be reluctant to alter existing clinical plans that are based on local interpretation. Perhaps not infrequently, the discrepancy is missed between the core laboratory and local interpretation. Finally, most open conversions have been performed under emergency circumstances where immediate control of the aorta is required, and so direct operative visualization before aortic cross-clamping has not been previously described.10 The operative identification of microleaks in patients A and D provided tremendous impetus for interpretation of previous studies and subsequent directed efforts to radiographically depict microleaks. Directed efforts typified in Figs 7 and 9 would not be typically performed in most surveillance protocols.

Techniques to identify microleaks are undergoing refinement and evaluation. Clearly, the standard CT and arteriographic protocols in widespread use are inadequate, although interpretation may improve with knowledge of these specific phenomena. The use of selective power injections within the iliac limbs, as demonstrated in patients B and D, raises concerns of artificially inducing transgraft flow. However, the arteriographic technique that was used was entirely routine, and would be safely applied to any iliac arterial circulation, regardless of native or artificial vascular conduit. Despite this, the careful use of hand injections with distal balloon occlusion, as in patient C, may be more appropriate.

Color flow duplex ultrasound scan is highly sensitive and is probably the best technique to identify microleaks if performed by skilled sonographers and properly interpreted. Doppler waveform assessment may also predict endoleak behavior.13 However, duplex scan is of limited utility in the obese patient. Other limitations include the possibility that motion of the endograft and adjacent thrombus within the AAA sac might produce similar color flow findings, mimicking actual blood flow outside the endograft. Doppler interrogation should help differentiate these phenomena (Fig 1). Certainly, visualizing small jets of color flow on opposite sides of an iliac limb suggests microleak versus motion artifact.

Type III transgraft endoleaks are not a new finding in endoprostheses and have been identified with other polyester fabrics, possibly because of chronic wear against metallic stents or calcified aortic plaque.14 Material fatigue has been identified at seams and sutures with other endovascular grafts.15,16 Microleaks with this endoprosthesis may be related to suture holes vacated by broken sutures. A second possibility is that balloon dilation during placement of the endograft causes fabric weave deformation resulting in gaps between the fibers. Another possible explanation for microleaks is failure of thin-walled graft fabric related to prolonged exposure to chronic pulsatile forces. It is not known if the separation of the stent from the graft material also results in increased fabric wear and predisposes to microleak. Combinations of multiple factors are likely.

All clinicians caring for patients treated with endovascular grafts must be made aware that transgraft endoleaks
may be currently underdetected and despite their small size can persist for more than 2 years. In fact, it is amazing that all microleaks do not spontaneously resolve. This may be related to the unique nature of intra-aneurysmal thrombus that has cellular elements and a continuous canalicular network and may explain the peculiar finding of grossly unusual thrombus seen during open conversion (patient A). Furthermore, even very small endoleaks may pressurize the aneurysm sac to systolic arterial levels, a finding that has been noted by others. It is concerning that the three patients followed up for more than a year all had striking AAA enlargement. Vascular surgeons and interventionists should continue to be involved in the care of these patients through the lifelong follow-up, because they are more familiar with failure modes of endovascular treatment than many primary care physicians.

Clearly, continued investigation of microleaks and their association with aneurysm sac pressurization and expansion is necessary. Microleaks may be primary causes of AAA enlargement, permissive factors with other endoleaks, or incidental, inconsequential findings. It is essential to rapidly determine what are the ideal diagnostic tests, the overall incidence of microleaks, and the clinical consequences of transgraft flow. The manufacturer of this particular endograft has formed a task force composed of external experts to address these important questions, and their findings are eagerly anticipated.

We thank Sara Minton for assistance with preparation of this manuscript. We are also grateful for comments and suggestions from Robert B. Rutherford MD, Christopher K. Zarins, MD, Rodney A. White, MD, William H. Pearce, MD, and James S. T. Yao, MD, Ph D.

REFERENCES


Please see related commentary by Dr James May on pages 369-70.