Relationship between preoperative patency of the inferior mesenteric artery and subsequent occurrence of type II endoleak in patients undergoing endovascular repair of abdominal aortic aneurysms

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Objectives: **The purpose of this study was (1) to find out whether preoperative inferior mesenteric artery (IMA) patency (on radiographic imaging) predicts IMA-related endoleaks after endovascular repair of infrarenal abdominal aortic aneurysms, (2) to determine feasibility of measuring aneurysm sac pressures in patients with endoleaks, and (3) to report early evidence of effective endovascular obliteration of IMA endoleaks.**
Methods: **We studied 76 consecutive cases of infrarenal aortic aneurysms that were repaired with an endovascular approach (March 1998–April 1999).**
Results: **There were 13 (17%) endoleaks persistent 30 days after the procedure. Eleven (85%) of these 13 were IMA-related endoleaks, which were documented with selective superior mesenteric artery angiography. The preoperative finding (on computed tomographic scan) of a patent IMA does not always predict an IMA-related endoleak, but results in a statistically and clinically significant higher ratio of patients with IMA-related endoleaks in the immediate postoperative period (24% versus 3%, $P < .035$). In eight of the 11 patients with persistent IMA-related endoleaks, measurement of intraaneurysm sac pressures was possible, and six of these patients had systemic pressures within the excluded aneurysm sac. Nine (82%) of 11 IMA-related endoleaks were successfully obliterated by means of selective IMA embolization.**
Conclusions: **Many endoleaks are caused by a patent IMA, and this can result in persistence of systemic pressure within the aneurysm sac. The preoperative finding (on computed tomographic scan) of a patent IMA is a predictor of increased rates of IMA endoleaks, and IMA endoleaks can be successfully obliterated through endovascular procedures, after endovascular abdominal aortic aneurysm repair. (J Vasc Surg 2000;32:777-88.)**

Since first introduced, the endovascular repair of infrarenal aortic aneurysms has been gaining increasing acceptance as favorable early outcome reports increase in numbers and feasibility reports proliferate, which expand the anatomic indications for this approach.[1-8] Despite the strong enthusiasm for this new method,[9-12] the consistent findings of high postprocedure endoleak rates (up to 21%) appear to represent a real and poorly understood problem.[3,10,13-15] The data presented from the Endovascular Technologies and Medtronic trials at the Federal Drug Administration panel this summer indicated up to 48% endoleak rates on early computed tomographic (CT) scan follow-up. Although great concern has been expressed over persistent anchoring site (type I) endoleaks (particularly prox-

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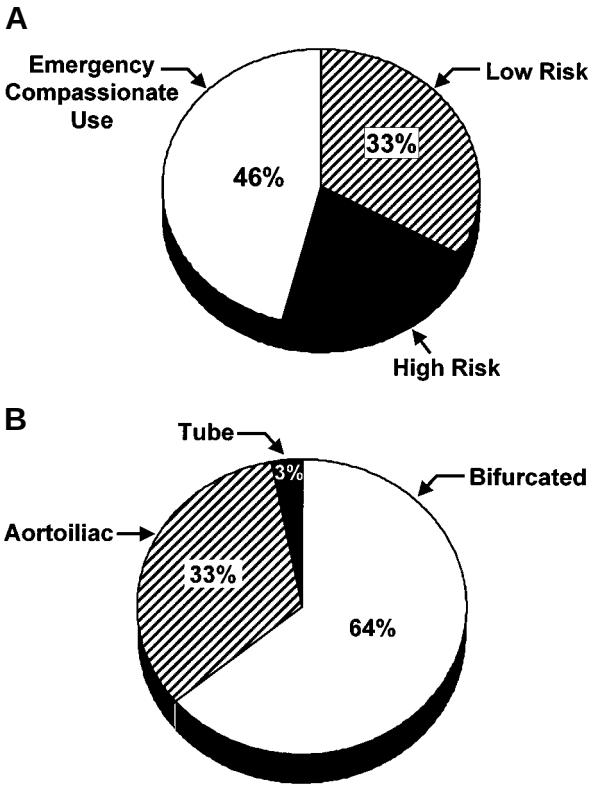

Fig 1. Diagrams showing the distribution of patients within the various clinical protocols: **(A)** indications and **(B)** graft designs.

imal endoleaks), little is known about the significance of type II endoleaks (related to the inferior mesenteric artery [IMA], lumbar arteries, and other collateral vessels).[16] A significant percentage of endoleaks (related to anchoring sites or collateral vessels) appears to spontaneously disappear after variable periods of follow-up.[3] Therefore, the time interval that defines a persistent endoleak that triggers concern varies widely among investigating centers. Even after the endoleak forms a thrombus and is no longer detectable on CT scan, it is unknown whether this occurrence eliminates transmitted pressure through the unexcluded thrombus to the aneurysm sac. If there remains transmitted systemic pressure to unexcluded aneurysmal aorta, one might anticipate that, analogous to an unrepaired thrombosed aneurysm, the risk of rupture would remain.

Because the risk of aneurysm rupture in different types of endoleaks is unknown, investigators at different centers have arbitrarily opted to follow up patients with different types of endoleaks for time intervals ranging from days to months to more than 1-year periods.[3] It has been assumed by many who are currently investigating this new minimally invasive procedure that only persistent proximal endoleaks carry the ongoing risk of aneurysm rupture. However, an increasing number of published and unpublished anecdotal reports indicate that patients may rupture their abdominal aortic aneurysms (AAAs) after what has been thought to be successful endovascular aneurysm exclusion, and even with documented aneurysm size reduction.[13,17] Not all reports are clearly connected to just proximal anchoring site endoleaks. For this main reason, careful long-term follow-up has been advocated by many, as we learn more about the effectiveness and durability of this new method of aneurysm repair.[16,18-23] Recently, an in vitro study suggested that even a small collateral vessel (0.410 mm) could maintain systemic pressure within an excluded aneurysm sac model.[24]

In this study, we focused on IMA-related type II endoleaks. Our goal was to begin to characterize the incidence and clinical significance of IMA-related type II endoleaks. We tried to answer specific questions of relevance to both doctors and patients: (1) To what extent does preoperative IMA patency, on radiographic imaging, represent a predictor of postoperative IMA endoleaks after endovascular repair of infrarenal aortic aneurysms? (2) Can an IMA endoleak transmit systemic pressure to the aneurysm sac? (3) Can an IMA endoleak be safely and successfully obliterated through the endovascular approach after primary stent grafting? (4) Alternatively, is there reason to attempt preoperative IMA embolization, when the vessel is seen to be patent by preoperative imaging modalities?

METHODS

Patients. The current work is a retrospective analysis of prospectively collected data that included our first consecutive 76 cases performed at our center from March 1998 to April 1999 in patients who underwent endovascular repair of infrarenal AAAs as part of an ongoing phase II multicenter clinical trial. All patients were enrolled after written informed consent was obtained in accordance with the guidelines and approved by our Institution Review Board and the Federal Drug Administration.

Indications and graft designs. As depicted in Fig 1, *A*, 46% of patients were treated as part of an "emergency compassionate" group. These were patients with a severe medical comorbidity that precluded open repair; they also had unfavorable aneurysm anatomy that disqualified them for enrollment in the established low- and high-risk standard protocols. Unfavorable

Table I. IMA patency by preoperative angiography versus postoperative IMA endoleaks

	IMA endoleak	*No endoleak*	*Total**
IMA patent by angiography	5	16	21
IMA not patent by angiography	5	43	48
Total†	10	59	69
% of patients with preoperative patent IMA by angiography	50%	27%	
χ^2 Test		$P = .279$	

*Two patients with anchoring site endoleaks were excluded.
†Five patients not studied by angiography preoperatively were excluded. (One patient with IMA endoleak and four patients without IMA endoleak.)

aneurysm anatomy included infrarenal aortic aneurysm neck length less than 15 mm, angulated neck greater than 45 degrees, trapezoidal or conical necks, dilated necks (> 28 mm), and narrow and tortuous iliac arteries that required a surgical conduit for access or required extensive angioplasty before access. Thirty-three percent and 21% of patients were enrolled in the low- and high-risk protocols, respectively. These patients' medical comorbidity was acceptable for potential open repair only in the low-risk group, but not in the high-risk group. Both the low- and high-risk standard protocols only included patients whose aneurysm anatomy was favorable for endovascular repair. The anatomy and extent of the aneurysm dictated the type of repair, after a specific philosophy that aims at complete exclusion of flow through aneurysmal parts of the aorta, the iliac arteries, or both. When aorto–uni-iliac devices were used, the clinical protocols required embolization of the contralateral common iliac artery (and ipsilateral hypogastric artery, depending on aneurysm extent). When the distal anchoring site was the external iliac artery, we adopted a protocol of preoperative embolization of the ipsilateral hypogastric artery. The rationale for this approach was to eliminate any potential type II endoleak that may be related to flow from the hypogastric artery, around the distal anchoring site and back into the aneurysm sac, with outflow through sac collaterals such as lumbar arteries or the IMA. In general, such planned preoperative embolizations were performed as separate staged procedures. The types of endoprostheses implanted are summarized in Fig 1, *B*, and included 59 modular (Talent; Medtronic/World Medical Manufacturing Corp, Sunrise, Fla) devices and 17 unitary (Ancure; Guidant/Endovascular Technologies, Menlo Park, Calif) devices.

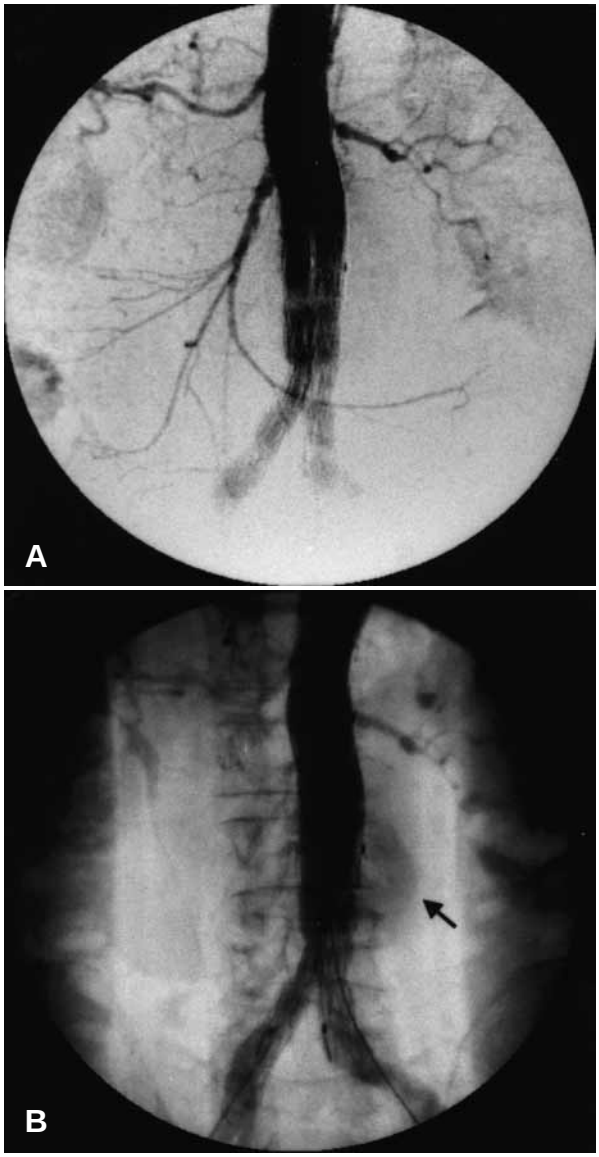

Fig 2. Intraoperative completion arteriograms showing **(A)** no evidence of endoleak and **(B)** evidence of an endoleak.

Preoperative determination of IMA patency. IMA patency was defined as visualization of this vessel in communication with the aneurysm as seen by use of preoperative imaging, which included a contrast CT angiography, conventional contrast arteriogram, and in selected patients (those with chronic renal insufficiency), magnetic resonance angiography (MRA). Diagnostic magnetic resonance imaging and MRA substituted CT scan/contrast arteriogram in five patients because of an elevated creatinine level. All radiographic studies were retrieved

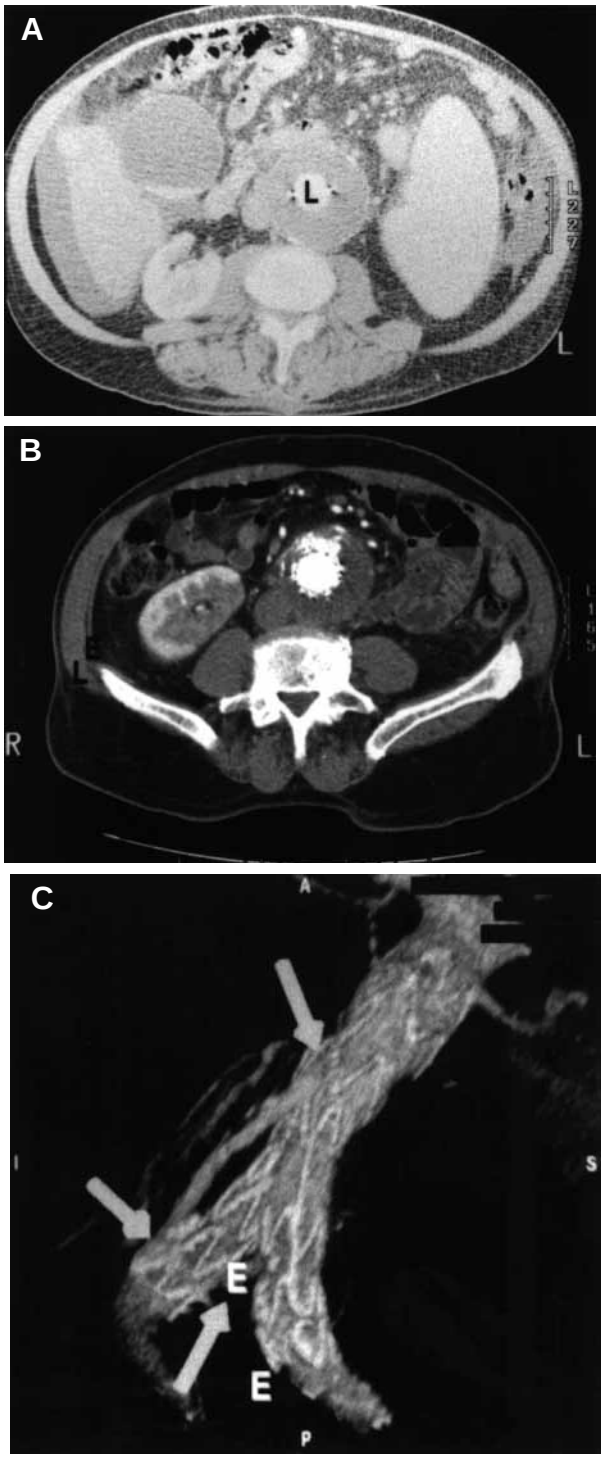

Fig 3. Postoperative CT scan showing **(A)** no evidence of endoleak, **(B)** evidence of endoleak of unspecified etiology (selective angiogram demonstrated this patient to have an IMA-related endoleak), and **(C)** an anchoring site endoleak identified by helical 3-D reconstruction CT angiogram. *E,* Endoleak; *L,* lumen.

and independently rereviewed in a blinded fashion by an expert radiologist.

Assessment for endoleaks. In the operating room after the device was implanted, the proximal and distal anchoring sites were individually studied with the use of antegrade and retrograde arteriograms to identify anchoring site endoleaks. If such an anchoring site problem was identified, further ballooning, stenting, or adding of covered extensions was performed until the operating surgeon was thoroughly satisfied that an anchoring site endoleak was completely ruled out. At this point, a power injection completion antegrade arteriogram, with delayed imaging, was performed. An endoleak was defined as any visualized contrast (including faint blush) filling the aneurysm sac outside of the stent graft (Fig 2). Postoperative CT scans (150 cc of iodinated contrast material at 4 mL/s with spiral 3-mm cuts with a pitch of 2) were performed before discharge and at 1 month of follow-up. Views obtained from these CT scans included predynamic, dynamic, and delayed contrast images that were reviewed for the presence or absence of endoleaks, with both the printed films (Fig 3) and a three-dimensional (3-D) work station. Some patients were studied postoperatively with ultrasound scanning, which can be useful in documenting the presence or absence of an endoleak[19] as shown in Fig 4. However, we observed early in our experience that in many patients, the postoperative ultrasound scan study was suboptimal or inconclusive.

Determination of endoleak etiology. Further diagnostic and therapeutic arteriograms were performed in patients with 30-day persistent endoleaks, which were aimed at clearly identifying the source of the endoleak. In our experience, CT scan and ultrasound scan modalities were insufficient for accurately determining the type of endoleak present. Because most centers now agree that a type I endoleak may essentially represent an untreated AAA, it is our philosophy that in the setting of an endoleak that has persisted for 30 days, one needs to rule out the presence of a type I endoleak. With this rationale, selective and supraselective arteriograms were used to accurately diagnose the source of all endoleaks noted to persist beyond 30 days after endovascular AAA repair. When technically feasible, this modality was also used therapeutically to obliterate endoleaks. These arteriograms were undertaken as a methodic and objective evaluation. First, an antegrade arteriogram by way of a proximally placed 5F pigtail catheter was performed; we specifically looked for proximal anchoring site endoleaks. Anterior-posterior and lateral intra-arterial

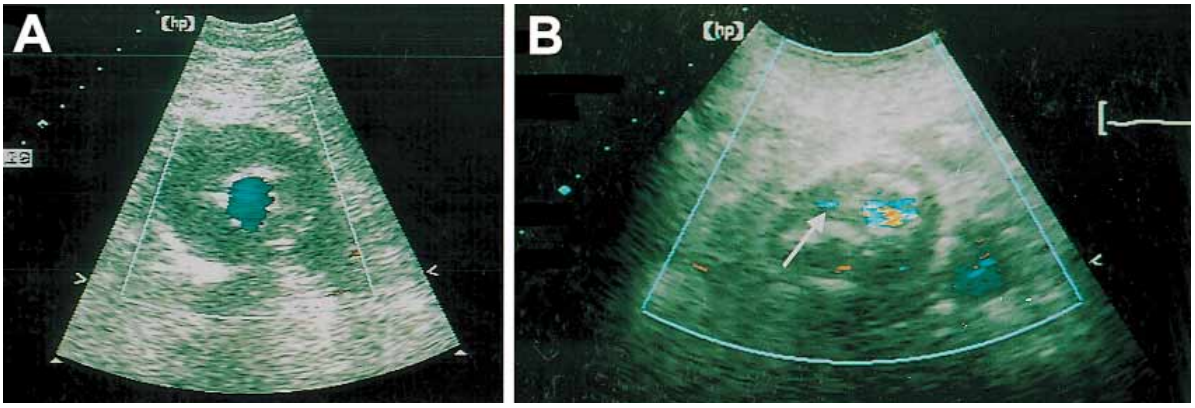

Fig 4. Postoperative ultrasound scans showing **(A)** no evidence of endoleak and **(B)** evidence of endoleak (*arrow*).

digital subtraction aortograms were performed with the pigtail positioned at the proximal stent graft anchoring site; we filmed at three frames per second and continued until contrast was completely washed out of the arterial and venous systems. Similar arteriograms were repeated with the pigtail moved to the graft-to-graft attachment sites. Retrograde arteriograms by way of distal femoral sheaths were then performed; we looked for distal anchoring site endoleaks. After anchoring site or graft-to-graft endoleaks were excluded, selective superior mesenteric artery (SMA) and hypogastric artery angiograms were performed; we looked for endoleaks related to collateral circulation (IMA or lumbar arteries, respectively). At this time, a microcatheter was advanced into the aneurysm sac (when technically feasible), and pressure measurements were obtained. Also at this time, selective embolization of the collateral vessel causing the endoleak was performed when feasible, as described below.

Treatment of type II endoleaks and measurement of aneurysm sac pressures. In the investigation and treatment of IMA-related endoleaks, a 4F or 5F catheter was placed at the proximal neck of the SMA, and an external coaxial tracker (3F, 150 cm) microcatheter (Fast Tracker; Boston Scientific, Natick, Mass) was used to cannulate the middle colic artery. The Tracker catheter was advanced through the middle colic and IMA into the aneurysm sac. After a hand-injected digital run was performed to confirm the origin of the endoleak, intrasac pressure measurements were made and compared with systemic arterial pressure. They were measured noninvasively at the level of the brachial arteries. The high resistance of the catheter system and the anatomic tortuosity of the collateral vessels en route to the IMA, in most cases, precluded measurement of systolic, diastolic, and pulse pressures. In those patients, mean pressure was therefore recorded for comparison with mean systemic pressure.

Table II. IMA patency by preoperative CT scan versus postoperative IMA endoleaks

	IMA endoleak	*No endoleak*	*Total**
IMA patent by CT	9	29	38
IMA not patent by CT	1	31	32
Total†	10	60	70
% of patients with preoperative patent IMA by CT	90%	48%	
χ^2 Test‡		$P = .035$	

*Two patients with anchoring site endoleaks were excluded.
†Four patients not studied by CT preoperatively were excluded.
‡Statistically significant difference ($P < .05$).

The microcatheter was then withdrawn to the proximal portion of the IMA, where microcoils were deployed until radiographic evidence of stasis of blood flow was obtained. Subsequent SMA arteriograms were performed to confirm elimination of the endoleak. Postembolization CT scans were also performed to examine the aneurysm sac and confirm the complete obliteration of the endoleak.

Statistical analysis. The relationship between patients who had postoperative IMA endoleaks and those who had evidence of a patent IMA by use of preoperative imaging was studied by calculating sensitivity, specificity, positive predictive value, negative predictive value, and accuracy for each of the preoperative imaging modalities alone and in combinations. Contingency tables were formulated and ana-

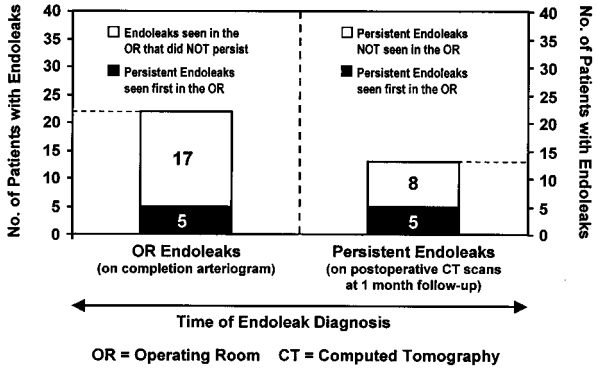

Fig 5. Graft indicating number of patients with endoleaks seen on completion arteriogram in the operating room versus those patients with 30-day persistent endoleaks visualized on follow-up CT scan.

lyzed with a χ^2 test. For these calculations, a 30-day persistent endoleak related to the IMA (as confirmed by selective SMA angiography) was defined as the gold standard of clinically significant IMA patency in this patient population. All calculations were performed with Primer of Biostatistics for Windows 95 version 4.0 (Stanton A Glantz; McGraw-Hill, Inc, Health Professions Division, Pittsburgh, Pa; part no. 864181-0).

RESULTS

There were 13 (17%), 30-day persistent endoleaks on postoperative CT scans at 1 month of follow-up. Only five of these were noted in the operating room on completion power injection arteriogram. The remaining eight were undetected on the operating room completion arteriogram. Conversely, 17 patients with evidence of endoleak on completion arteriogram in the operating room subsequently had no evidence of endoleak on follow-up CT scans (Fig 5).

With the use of a CT scan plus standard and selective angiography, 11 (85%) of 13 endoleaks persisting at 30 days were confirmed to be related to collateral circulation from the IMA into the aneurysm sac (Fig 6, *A*). Lumbar arteries and branches of the hypogastric arteries were often identified as sources of outflow by injecting contrast directly into the aneurysm sac through the same catheter used to measure aneurysm sac pressure (Fig 7). There were one proximal and one distal anchoring site endoleaks (2.6% overall, 15% of all endoleaks). During the course of this study, all patients with persistent flow through the aneurysm sac were noted to have patent lumbar arteries that served as egress vessels (including the two patients with type I endoleaks), but antegrade flow into the aneurysm sac from a lumbar artery source was not seen in this series of patients, despite thorough evaluation that included selective hypogastric artery arteriography. Also, anchoring site endoleaks were repaired with covered stent graft extensions as secondary endovascular procedures.

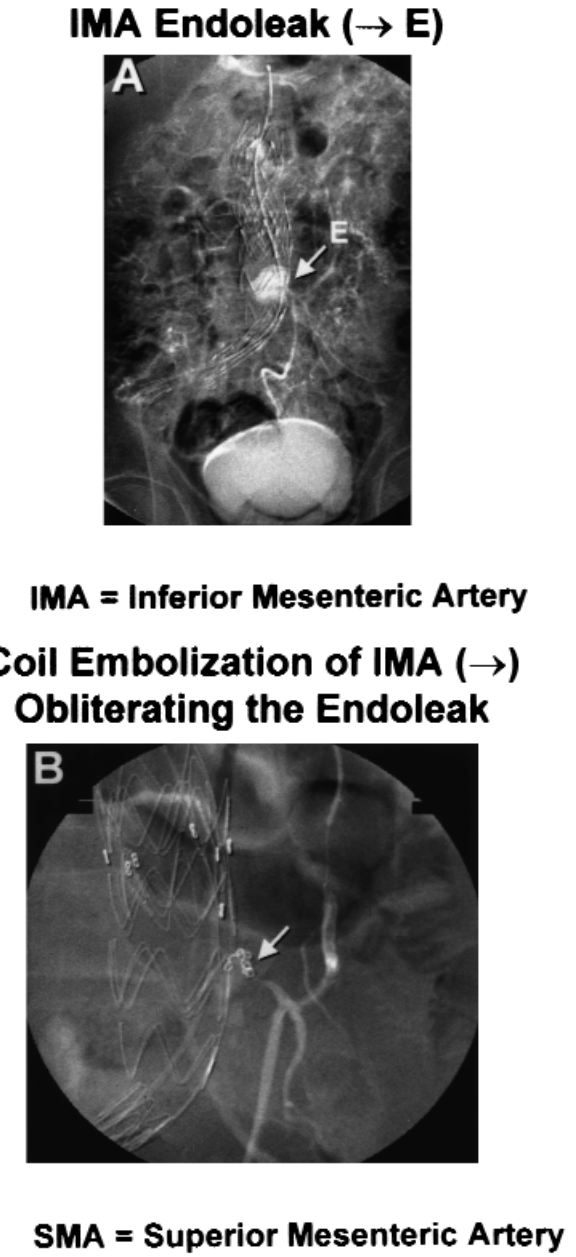

Fig 6. Postoperative selective SMA angiography for **(A)** diagnosis and **(B)** treatment of IMA endoleak. *Arrow* in **A** points to contrast blush into aneurysm sac immediately next to IMA, which fills retrograde through SMA collaterals. *Arrow* in **B** points to microcoils within the IMA, now obliterating the contrast blush. *E,* Endoleak.

Table III. Ability of preoperative study to predict postoperative IMA endoleak

	Preoperative study documenting IMA patency			
	CT	*Angiography*	*CT and angiography (concurring reading)*	*CT, angiography, or MRA (any preoperative imaging indicating IMA patency)*
Ability of preoperative study to predict postoperative IMA endoleak by				
Sensitivity (%)	90	50	50	91
Specificity (%)	52	73	81	41
Positive predictive value (%)	24	24	31	21
Negative predictive value (%)	97	90	91	96
Accuracy (%)	57	70	77	49

The IMA patency revealed through preoperative angiogram and preoperative CT scan in patients with and without postoperative IMA endoleaks is shown in Tables I and II. Angiography revealed a preoperative patent IMA in 50% of patients with an IMA-associated endoleak. Only 27% of those patients without an endoleak had an angiographically patent IMA. This difference did not reach statistical significance (Table I). Ninety percent of patients with an IMA-related endoleak had a preoperative patent IMA detected through CT scan versus only 48% of those without an endoleak (Table II). This difference was statistically significant. Twenty-four percent of patients with a patent IMA detected through preoperative CT scan versus only 3% of those without a patent IMA (detected through preoperative CT scan) had IMA-related type II endoleaks that persisted at 30 days after stent graft repair of their AAAs ($P < .05$).

The sensitivity, specificity, predictive value, and accuracy of the imaging modalities were calculated, with the assumption that a 30-day persistent IMA endoleak was the gold standard measure of a clinically relevant IMA patency, to determine whether one can predict postoperative IMA-related endoleaks on the basis of IMA patency revealed through preoperative imaging (Table III). Sensitivity was best for the CT scan alone at 90%. Specificity and accuracy were best for concurring readings on CT scan and angiogram. The negative predictive value was high with all single and combination imaging modalities (highest for CT scan alone at 97%; Table III). The positive predictive value and accuracy were uniformly low (Table III).

In eight of the 11 patients with an IMA-related 30-day persistent endoleak, the aneurysm sac pressure was measured with the use of a catheter technique as depicted in Fig 7 and Table IV. Six of these patients had systemic pressures within the aneurysm sac. In the remaining three patients, the measurement of sac pressure was not technically feasible because of vessel caliber and tortuosity, which resulted in an inability to advance the microcatheter.

Table IV. Intra-aneurysm sac pressure in patients with IMA endoleaks

Endoprosthesis design	*Intra-aneurysm sac pressure* compared with system pressure†*
Aortoiliac	Systemic
Aortoiliac	Systemic
Bifurcated	Systemic
Bifurcated	½ systemic
Bifurcated	Systemic
Aortoiliac	Systemic
Bifurcated	½ systemic
Bifurcated	Systemic

*Aneurysm sac pressures were measured with trans-IMA catheter pushed into the sac, a translumbar catheter pushed into the sac, or a catheter negotiated into sac directly between the proximal anchoring site and the aortic wall.
†Systemic pressures were measured at an upper extremity.

Selective embolization of the IMA was possible with subsequent obliteration of the IMA-related endoleaks in nine of 11 patients (Fig 6, *B*). In the other two patients, one endoleak thrombosed spontaneously at 1 month of follow-up, and one patient died of severe preexisting hepatic dysfunction. All patients with subselective embolization of the IMA had angiographic resolution of the endoleaks and negative 1-month follow-up CT scans (Fig 8).

In this series, all surviving patients are free of endoleaks; 13 (17%) of 76 patients have required secondary endovascular procedures aimed at obliterating 30-day persistent endoleaks. One patient (1%) had postoperative colonic ischemia that required colon resection. This patient had a history of colon resection, had a patent IMA revealed through preoperative imaging, and required an aortoiliac device for aneurysm repair. This patient also underwent staged

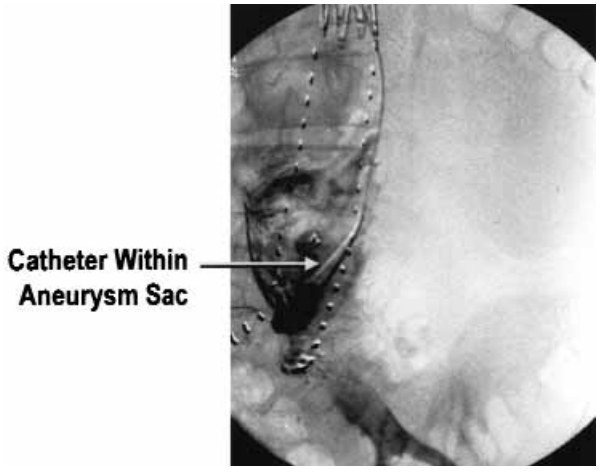

Fig 7. Radiograph showing catheter within the aneurysm sac during measurement of intra-aneurysm sac pressure after endovascular repair in a patient with a 30-day persistent endoleak.

embolization of both hypogastric arteries. A patent IMA without any evidence of postoperative colon ischemia was seen in three other patients repaired with aortoiliac devices; none had prior colon surgery.

DISCUSSION

The current work has shown that a significantly higher proportion of patients whose preoperative CT scan demonstrates a patent IMA had postoperative IMA-related endoleaks compared with those whose preoperative CT scan *did not show* a patent IMA (9 [24%] of 38 vs 1 [3%] of 32; $P < .05$). Similarly, a significantly higher proportion of patients with an IMA-related endoleak had a patent IMA visualized with preoperative CT scan compared with patients without an endoleak (9 [90%] of 10 vs 29 [48%] of 60; $P = .035$) (Table II). The preoperative identification of a patent IMA on arteriogram did not have a similar predictive value (Table III). Therefore, the preoperative identification of a patent IMA on CT scan was associated with a statistically and clinically significantly higher proportion of patients with postprocedural type II endoleak (which included inflow from the IMA and outflow through patent lumbar arteries).

In a clinical sense, however, a useful significant predictor of postoperative type II endoleak could discriminate which of the patients with a patent IMA would be most likely to have an IMA-related type II endoleak (ie, there would be a clinical test with high positive predictive value and high accuracy). An analysis of the preoperative imaging studies alone and in combination demonstrates that the positive predictive value and accuracy are not sufficiently high enough to discriminate clinically between patients in such groups; that is, in patients for whom the preoperative imaging indicates a patent IMA, most *will not* have an IMA-related endoleak (Table III). For these reasons, we do not advocate preoperative interventions (eg, embolization of the IMA) based on the preoperative imaging studies, because this would result in many unnecessary interventional procedures. This recommendation is further strengthened by our success with the postoperative endovascular obliteration of IMA endoleaks by means of selective embolization (in those patients with persistent endoleaks).

Although it might be considered intuitive or "self-evident" that patients with nonpatent IMAs should not have IMA-related endoleaks, it is reassuring that the data confirm this expected finding, while demonstrating the constraints of the overall incidence of false positives and false negatives and the global sensitivity, specificity, and accuracy of the preoperative imaging studies as they demonstrate the status of the IMA. Because radiographic imaging data depend on technical aspects of the performance of the study as well as the subjective interpretation of the radiologist, we believe that the statistical analysis performed in this study bears significant relevance to the question of whether a clinician can trust such data for the purposes of patient information, predictions, or both as to the potential trouble with IMA-related endoleak. Although it is true that fewer than a quarter of patients who had a radiographically patent IMA went on to have an IMA-related endoleak, it is also true that a statistically and clinically significant higher ratio of patients with a patent IMA (radiographically) went on to have an IMA endoleak when compared with the group of patients who had a radiographically nonpatent IMA.

We have also identified in this series that intraoperative endoleaks visualized on completion arteriogram are not necessarily predictive of postoperative persistent endoleaks visualized on follow-up CT scans. Because the standard nonselective aortogram does not appear to be the best method for detecting endoleaks and because many collateral branches may thrombose spontaneously between the completion arteriogram and the first postoperative CT scan, we were not surprised to find little correlation between the endoleaks seen in the operating room completion arteriogram and those seen on postoperative CT scans.

With the measurements of aneurysm sac pressure in patients who had IMA-related endoleaks, we have

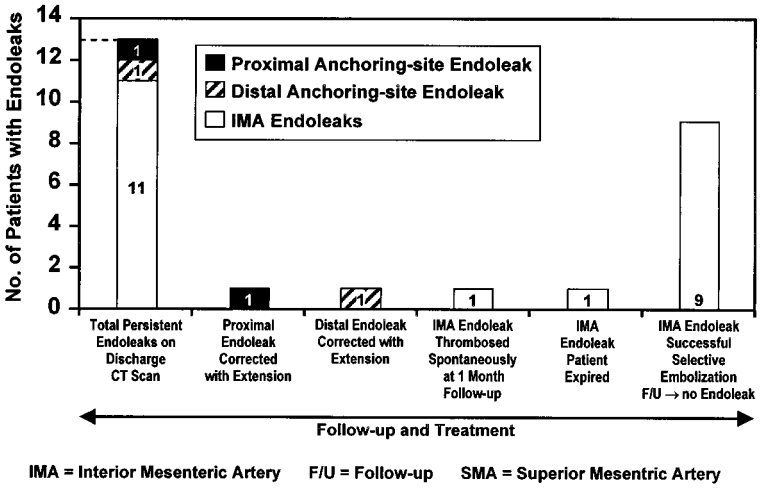

Fig 8. Summary of the follow-up and treatment in all patients with 30-day endoleaks.

established feasibility of this technique and demonstrated that a significant number of patients with an IMA endoleak continue to have systemic pressure within the aneurysm sac. The presence of the microcatheter within the IMA may actually lead to alterations in the detected pressure within the aneurysm sac (by virtue of the microcatheter partially occluding flow within the patent IMA). We believe that this may in fact account for the lower pressure recorded in the two patients and the overall decreased pulse pressure noted within the aneurysm sac. However, this is only speculation, and the data available are insufficient to determine with certainty what might be the etiology or clinical significance of the measured pressures. Further studies are clearly warranted on this subject. Moreover, in view of the fact that intrasac pressures were not measured in any patient in whom there was not a postoperative endoleak, one cannot be absolutely certain that the patent IMA is the only cause of the high intrasac pressure. It is unlikely that the high intrasac pressures were maintained by patent lumbar arteries in these studied patients, given the angiographic evidence, as discussed later. Alternatively, it might be possible that the pressure may be actually transmitted through the fabric wall into the aneurysm sac. The current study was not designed to answer these difficult questions, which would require the measurement of sac pressures in patients without an endoleak and with various types of endoleaks. The current study represents the first feasibility report. Further studies designed to answer these important physiologic questions are certainly indicated.

In this series we have noted an overall 30-day persistent endoleak rate of 17%, which is within the range of what others have reported,[3,5,9,25-29] but we have a preponderance of type II endoleaks, all of which have been IMA-related endoleaks (IMA identified as inflow, lumbar arteries visualized and likely to represent outflow, as discussed below). We would have expected a higher rate of anchoring site endoleaks, given that 46% of patients enrolled had extreme unfavorable anatomy for endovascular repair, but this was not seen possibly because of our preference for suprarenal fixation and the preponderant use of modular devices that allow for easy intraoperative repair of detected anchoring site leaks. This distribution of endoleak etiology has not been frequently noted by other investigators. One may postulate that such a difference reflects a sampling variability related to a different set of patients with a differing range of aneurysm anatomic features, to differences in the types of endoprosthesis used, or to differences in fixation choice (most of our patients were repaired with suprarenal bare-spring fixation). However, it is more likely that our methodic and extensive study designs aimed at clearly identifying the exact endoleak inflow source account for our findings. This rigorous, objective approach that we have pursued for the identification of endoleak etiology has not been previously undertaken. Most earlier reports have not used postoperative selective angiography in the pursuit of endoleak etiology. Most authors have relied on postoperative CT scans and ultrasound scanning in the evaluation of postoperative endoleaks.[3,18-23]

In many patients, the anatomic variability of where the collateral vessels enter and exit the

aneurysm sac precludes an exact localization of the endoleak inflow source when only CT or ultrasound scan imaging is used, even with the use of dynamic spiral CT angiography with 3-D reconstruction; however, in one patient we were able to clearly demonstrate the endoleak source using this latter modality (Fig 3, *C*). Standard antegrade anterior-posterior and lateral aortography was also limited in many cases, in terms of clearly localizing the source of endoleaks. One case (a proximal endoleak) was diagnosed by the use of this latter modality without need for further selective images. However, most cases required selective angiography to clearly identify the endoleak inflow source. In some patients with CT scan evidence of an endoleak, no obvious contrast blush was noted with standard aortography. In these cases, selective arteriograms revealed the small but definite contrast extravasation into the aneurysm sac. In all cases, a source of egress from the sac was noted through small lumbar or hypogastric artery branches.

In our series, lumbar arteries, branches from the hypogastric arteries, or accessory renal arteries were not seen to fill the aneurysm sac antegrade as a primary source of a type II endoleak. Because preoperative angiogram and CT scan showed these branches to be patent in many patients, we believe they were small enough to have thrombosed after stent grafting. When patent, they did not show antegrade flow into the aneurysm sac but appeared to serve as egress vessels. Similarly, many patients with a patent IMA revealed through preoperative imaging did not show an IMA-related endoleak, presumably because of thromboses of this vessel after stent grafting.

In all cases where the IMA produced an antegrade contrast blush in the aneurysm sac, lumbar artery branches were identified as egress vessels on delay filming. As a matter of traditional radiographic convention, we have attributed the cause of the endoleak to the source of inflow (in our series, the IMA). Other groups have reported a higher rate of lumbar artery–related type II endoleaks. Thus, one may question whether some of the endoleaks observed in our series have inflow through the lumbar arteries. However, the selective hypogastric arteriograms fail to indicate antegrade flow from lumbar artery branches in the aneurysm sac. The type II endoleaks noted were identified only on selective SMA and IMA arteriogram. It would be unlikely that contrast dye injected under pressure through a catheter located in the IMA would diffuse through the aneurysm sac and then flow against systemic pressure into the lumbar arteries. This contrast would then follow retrograde, against systemic pressure, into the patent lumbar arteries and drain through the venous system only if the lumbar arteries represented a low-pressure system acting as an outflow for the endoleak. We are certain that the collective data obtained from the thorough selective and subselective arteriography were sufficient to precisely identify the nature and source of the endoleak at hand in each patient. Therefore, it was concluded that in the series reported, the type II endoleaks observed thus far show inflow from the IMA and outflow through the lumbar arteries. To the extent that flow through an endoleak mandates both inflow and outflow (from a high-pressure system to a low-pressure system), the patent lumbar arteries contributed to the endoleak. However, selective embolization of the IMA has resulted in successful obliteration of the endoleak (with angiography and CT scan) in all cases thus far.

The patient who had colonic ischemia had a patent IMA preoperatively revealed through CT scan and angiogram. Stent grafting resulted in thrombosis of this vessel, which for this patient must have been essential for distal colon perfusion because his prior colon surgery compromised other natural collaterals. The endovascular approach precludes the intraoperative decision of reimplanting the IMA on the basis of direct colon inspection, and therefore, such decision making becomes part of the preoperative planning. For this reason, evaluation of the preoperative imaging modalities with which one looks for IMA patency plus consideration of any prior colon surgery becomes essential in determining stent graft design and feasibility for the endovascular approach. This is becoming increasingly important as the current enthusiasm for expanding the applications for this new technology continues to grow.[6-8,21,23,30]

Although most agree that anchoring site endoleaks represent a failure to successfully repair the aneurysm with the use of the endovascular method, the significance of type II endoleaks remains unknown, and there is no consensus for a standard treatment.[3,30-33] Some have reported success with embolization of the feeding collateral vessel responsible for the endoleak,[3,33] whereas others have advocated embolization of the aneurysm sac itself.[26,32] This latter method continues to carry the question of whether systemic pressure can be transmitted through a clot to the aneurysmal aortic wall. In some cases the option has been to observe without treatment depending on feasibility and success of secondary procedures.[3] In Zarins et al's report,[3] 9% of patients continued to have an endoleak at 6 months

of follow-up, but all endoleaks were no longer seen on CT scan at 15 months of follow-up. They report no aneurysm ruptures during follow-up and evidence of aneurysm size decreasing from 6.5 to 5.0 cm in one patient at 1 year of follow-up despite persistence of the endoleak. Other authors have also noted a lack of aneurysm growth on short-term follow-up despite persistent endoleaks.[34] However, it is difficult to interpret what this means for a patient, because some authors report aneurysm rupture despite successful stent grafting and aneurysm rupture in the presence of aneurysm size decreases.[13,17,18,35] Is it possible that, to some degree, we are rediscovering the natural history of untreated or partially treated AAAs?

In our series, one patient who had a symptomatic aneurysm (pain and tenderness) continued to have symptoms until a type II endoleak was successfully treated by coil embolization of the IMA. Another patient with an endoleak (of unknown etiology) at discharge died suddenly after collapsing with abdominal pain at home, before the 1-month follow-up CT scan. This patient had a 7.5-cm aneurysm with minimal thrombus within the sac. Schurink et al's[24] in vitro model elegantly demonstrated that every endoleak, even a very small one, caused more than systemic diastolic pressure within the aneurysm sac. Our data have shown that IMA endoleaks can result in systemic pressure within the aneurysm sac. For these reasons we have chosen the approach that the aneurysm is not completely repaired until the patient is free of endoleaks. Recognizing that it may take days before all the small collaterals thrombose and knowing that, in fact, a significant number do thrombose at 30 days of follow-up, we have chosen 1 month as our follow-up interval before elective secondary endovascular procedures aimed at diagnosis and potential treatment. Our primary concern and clinical rationale for the 30-day selective arteriogram for persistent endoleaks were any abilities to rule out anchoring site endoleaks. Our premise has been that anchoring site endoleaks may behave like untreated AAAs. Thirty days represent an arbitrary interval that lies within what one might consider to be a reasonable waiting period for elective open repair (to date, the standard method of treatment). However, one might stipulate, on the basis of our experience, that this interval may need to be shortened in patients with symptomatic aneurysms or those with large aneurysms and little thrombus within the aneurysm sac. Others may contend that this interval is too aggressively short and may choose to follow up these patients for longer periods, provided the aneurysm does not progressively expand.

In summary, a significant number of type II endoleaks are caused by a patent IMA. IMA endoleaks can transmit systemic pressure to the aneurysm sac. Selective SMA angiography appears to be the most effective method to identify IMA endoleaks. The preoperative CT scan can be useful in predicting the risk of IMA-related endoleaks, but its accuracy and positive predictive value are not sufficiently high enough to advocate preemptive IMA embolization. Selective coil embolization of the IMA can safely and successfully obliterate persistent IMA endoleaks after aortic stent grafting. The physiology and clinical significance of endoleaks needs further study.

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