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Evaluation and Treatment of Suspected Type II Endoleaks in Patients with Enlarging Abdominal Aortic Aneurysms

Brian Funaki, MD, Nour Birouti, MD, Steven M. Zangan, MD, Thuong G. Van Ha, MD, Jonathan M. Lorenz, MD, Rakesh Navuluri, MD, Christopher L. Skelly, MD, and Jeffrey A. Leef, MD

Section of Vascular and Interventional Radiology, Department of Radiology (B.F., N.B., S.M.Z., T.G.V.H., J.M.L., R.N., J.A.L.), and Department of Surgery (C.L.S), University of Chicago Medical Center, 5840 S. Maryland Ave., MC 2026, Chicago, IL 60637

Abstract

Purpose—To evaluate angiographic diagnosis and embolotherapy of patients with enlarging abdominal aortic aneurysms and computed tomographic (CT) diagnosis of type II endoleak.

Materials and Methods—A retrospective review was performed of all patients referred to a single vascular and interventional radiology section from January 1, 2003, to June 1, 2011, with a diagnosis of enlarging aneurysm and type II endoleak. Twenty-five patients underwent 40 procedures between 12 and 82 months after endograft insertion (mean, 48 mo) for diagnosis and/or treatment of endoleaks.

Results—Type II endoleaks were treated with cyanoacrylate, coils, and ethylene vinyl alcohol copolymer in 16 patients. Technical success rate was 88% (14 of 16 patients) and clinical success rate was 100% (16 of 16 patients). Aneurysm growth was arrested in all cases over a mean follow-up of 27.5 months (range, 6 – 88 mo). Endoleaks in nine patients were misclassified on CT; two had type I endoleaks and seven had type III endoleaks. Four of the nine patients (two type I endoleaks and two type III endoleaks) were correctly classified after initial angiography. The other five type III endoleaks were correctly classified on CT after coil embolization of the inferior mesenteric artery. Direct embolization was performed via sac puncture with ethylene vinyl alcohol copolymer in two of the latter five patients and eliminated endoleaks in both.

Conclusions—Aneurysm growth caused by type II endoleaks was arrested by embolization. CT misclassification occurred relatively commonly; type III endoleaks purported to be type II endoleaks were found in 28% of patients (seven of 25).

Type II or side branch endoleaks are the most common complication of endovascular aneurysm repair. Although they are usually benign, some type II endoleaks are associated with sac enlargement and occasional rupture (1,2). There is a consensus that type II endoleaks should not be treated in the absence of aneurysm growth (2), but reported experience with embolotherapy consists of heterogeneous series of patients with stable and enlarging aneurysms (3–10). Because the patient populations of these reports have not been stratified, the results of embolization of type II endoleaks are difficult to interpret in patients with expanding aneurysms (3–10). The purpose of the present investigation was to examine the outcomes of the patients who were referred to our vascular and interventional radiology

section for evaluation and treatment of type II endoleaks associated with enlarging aortic aneurysms.

MATERIALS AND METHODS

This was a retrospective observational study conducted at a university medical center and was approved by our institutional review board. We reviewed all patients referred to our section from January 1, 2003, to June 1, 2011, with diagnosis of type II endoleak by computed tomography (CT) and proven aneurysm enlargement after endovascular aneurysm repair (Table). Endoleaks were diagnosed from 12 to 82 months (mean, 48 mo) after aneurysm repair; the date of endograft repair was not available in four patients, although, based on patient history, these patients had grafts placed more than 1 year before referral. One patient who had undergone previous embolizations at several outside institutions was excluded from analysis. A total of 40 procedures were performed in 25 patients (five women and 20 men; age range, 60–92 y; mean age, 80 y) referred from multiple hospitals in three states. The types of endografts and number of patients with each type of endograft included EXCLUDER (W.L. Gore and Associates, Flagstaff, Arizona; n = 11), Zenith (Cook, Bloomington, Indiana; n = 4), Ancure (EVT/Guidant, Sunnyvale, California; n = 3), and AneuRx (Medtronic, Santa Rosa, California; n = 7) devices. All endoleaks were diagnosed with CT; sac size was measured at largest diameter on axial images. Sac enlargement was defined as an increase of more than 5 mm in sac diameter compared with immediate postprocedure CT. CT scans varied in terms of scanner age and techniques used for evaluation of endoleaks. The qualifications of interpreters were unclear but reflected the gamut of clinical practice within our hospital and the greater community. At least two CT studies (including one immediately after endografting) were reviewed before angiography to confirm aneurysm growth and presence of endoleak. Copies of follow-up postembolization CT studies obtained at outside hospitals were sent to our section and reviewed during follow-up. We had no information regarding the overall number of endografts placed by the various physicians who referred these patients to our section.

Our staged management algorithm is given in Figure 1. Briefly, after review of cross-sectional imaging, a trans-femoral angiogram was obtained. Aortography in frontal and lateral positions was performed, followed by selective catheterization of the ascending lumbar arteries or inferior mesenteric artery (IMA) via the superior mesenteric artery. If a type I or III endoleak was visualized on aortography, embolization was not performed. Instead, patients were treated with additional grafts to exclude the endoleak or underwent open repair. These procedures were not performed in our interventional radiology section; instead, patients were sent back to their original referring physicians for these procedures. If the IMA was patent and communicated with the endoleak, the IMA origin and/or endoleak was embolized with coils. A repeat CT examination was performed at 1 month. If the endoleak persisted, a additional embolization was performed via transarterial approach or translumbar approach. The goal of embolization was occlusion of the endoleak cavity within the aneurysm sac, but, if the cavity was not accessible, coil embolization of all communicating arteries was pursued instead. Technical success was defined as successful embolization with no further filling of the endoleak cavity on angiography and follow-up CT. Clinical success was defined as regression or stabilization of the aneurysm sac (ie, shrinkage or no further growth) irrespective of endoleak cavity filling during continued follow-up (minimum 6 mo follow-up). Patients were followed with serial 6-month CT examinations after completion of staged therapy.

Transarterial Embolization

To perform embolization, the left or right common femoral artery was punctured and a 5-F sheath was inserted. Depending on the location of the suspected endoleak, under

fluoroscopic guidance, coaxial technique (5-F VS-1 [Cook] and 2.7-F Progreat [Terumo, Somerset, New Jersey]) was used to catheterize the ascending lumbar arteries via the hypogastric artery or the IMA via the superior mesenteric artery (middle ascending left colic arcade or arc of Rioloan). When possible, the portion of the endoleak within the aneurysm sac was catheterized and an angiogram from within the sac was obtained. Embolization was performed within the sac when possible. For the first 13 patients, microcoils (fibred platinum coils; Boston Scientific, Natick, Massachusetts) and/or cyanoacrylate glue (Trufill; Cordis, Miami Lakes, Florida) were used. For the latter 13 procedures, ethylene alcohol copolymer (Onyx-18; ev3, Plymouth, Minnesota) was used instead of glue. For all the cases that involved cyanoacrylate glue, a 3:1 mixture was injected through the microcatheter and the microcatheter was pulled. Ethylene alcohol copolymer was injected similarly, with the catheter being pulled after the cavity within the aortic sac was filled. Liquid embolic agents were used only in patients in whom the sac could be catheterized and in whom the IMA had been coil-embolized or was not patent.

Direct Aortic Access

To achieve aortic access, the patient was positioned in a supine or prone position, and, by using CT guidance, the endoleak cavity was punctured with a 21-gauge needle (AccuStick set; Boston Scientific). After blood return, the 0.018-inch guide wire included in the kit was advanced into the leak, followed by a 4-F dilator (ie, the 6-F outer dilator of the AccuStick set was removed and the 4-F dilator and inner metal stiffener were advanced over the 0.018-inch guide wire). Subsequently, the patient was moved from the CT scanner to the interventional radiology suite and embolization was performed under direct fluoroscopic guidance. A liquid embolic agent (cyanoacrylate or ethylene alcohol copolymer as noted earlier) was chosen in an attempt to fill the endoleak completely and slightly reflux into all communicating arteries. As noted earlier, the catheter was pulled after embolization.

RESULTS

Treatment of Confirmed Type II Endoleaks

Type II endoleaks were confirmed and treated in 16 patients. Twenty-eight procedures were performed in this group to complete treatment. The following embolic agents were used: coils (n = 21 procedures), cyanoacrylate glue (n = 3 procedures), and ethylene vinyl alcohol copolymer (n = 4 procedures). Technical success was achieved in 14 of 16 patients (88%). In two of the technical failures, persistent endoleaks were observed after embolization. In both cases, qualitatively, endoleaks within the aortic sacs decreased in size on follow-up CT scans. Both endoleaks were located anterior to the endograft, and direct percutaneous access was considered technically difficult or relatively contraindicated as a result of the closely apposed adjacent bowel. It was our plan to attempt direct puncture if aneurysms continued to enlarge, but aneurysm growth was arrested. Both patients have had stable aneurysms at 67 and 72 months of follow-up.

Clinical Outcomes

Clinical success was achieved in all patients with type II endoleaks who underwent embolization, irrespective of technical success. All had stable or shrinking aneurysm sacs at a mean follow-up of 27.5 months (range, 6 – 88 mo). Two patients were lost to follow-up after 1 year—both had stable aneurysms at that point in time. Two separate patients developed new type II endoleaks after successful embolization. Both were followed by serial imaging, and aneurysm sac size remained unchanged. There were no ruptures. An 85-year-old man with a Medtronic endograft who underwent a technically successful procedure developed a type III endoleak 8 months later, which was manifest by complete detachment

of an aortic cuff from the main body of the graft shown on an abdominal radiograph. He underwent open repair.

Complications

Per Society of Interventional Radiology clinical practice guidelines (11), there were two minor procedure-related complications. One patient developed a moderate-sized groin hematoma after transarterial embolization and a second patient developed a small retroperitoneal hematoma after translumbar embolization. Both were self-limiting and resolved without further therapy.

Misclassified Endoleaks

Nine endoleaks purported to be type II endoleaks were misclassified. Two were type I endoleaks and seven were type III endoleaks (type IIIa, $n = 2$; type IIIb, $n = 5$). Graft components had not separated on type IIIa junctional leaks. Patients with type III endoleaks were treated with the following grafts: EXCLUDER ($n = 3$), AneuRx ($n = 3$), and Ancure ($n = 1$). Both type I endoleaks and two of the type III endoleaks were discovered during initial angiography and embolization was not performed. Instead, these patients returned to their referring physicians at the referring physician's request for repeat stent-graft insertion. All these patients were referred from outside hospitals, with CT scans performed in the portal venous phase only. CT scans in all four cases showed a large endoleak with enhancement of the IMA and/or lumbar arteries. All were interpreted as representing type II endoleaks by radiologists external to our institution. Five of the type III leaks were correctly diagnosed only after embolization when the leak was still visualized on CT after occluding outflow vessels. All five of these patients had arterial- and venous-phase CT examinations. Two of these five patients had their type III leaks successfully treated by filling the portion of the endoleak within the aortic sac with ethylene vinyl copolymer. Both patients had stable aneurysms on 6- and 9-month follow-up. The other three patients were discovered to have type III endoleaks after collateral branches were embolized and endoleaks persisted on CT. These three patients underwent repeat stent-graft deployment to cover the graft rent or conversion to aortouniiliac grafts with femoral-to-femoral artery bypass grafts.

DISCUSSION

Currently, there is general agreement that type II endoleaks should be treated only in patients with enlarging aneurysms (1,2). However, all previously reported series of type II endoleak embolization included patients with stable and enlarging aneurysms (3–10). Whether results for patients with growing aneurysms are different from those with stable aneurysms is unclear. Because many type II endoleaks will resolve spontaneously and many others that persist do not lead to adverse outcomes, treating all endoleaks may spuriously improve the outcomes (from an overall percentage standpoint) of embolotherapy compared with treating only enlarging aneurysms. Therefore, although series published to date have established the safety of embolotherapy for all type II endoleaks, it is difficult to draw firm conclusions regarding clinical efficacy of embolotherapy for growing aneurysms. The present series describes results of embolization in patients with proven aneurysm enlargement and type II endoleaks classified by CT. Our experience suggests that this group is unusual in that a relatively high percentage of these patients had their endoleaks misclassified. It is important to note that misdiagnosis in this series may be attributed to a number of factors, including inconsistent CT imaging and interpretation. Misclassified type III endoleaks rather than type II leaks led to aneurysm expansion in more than one fourth of the patients in the present study.

Because of the differences of our patient population compared with those in reports (ie, we treated only patients with enlarging aneurysms whereas earlier investigators treated patients with enlarging and stable aneurysms), we defined two endpoints. Technical success was defined as obliteration of the endoleak and clinical success was defined as stabilization of the aneurysm sac. We believe that, in a patient with a growing aneurysm, arresting growth and preventing rupture are the best indicators of clinical success—not the presence or absence of an endoleak on CT. Our technical success was similar to the that from largest reported series (9), in which Stavropoulos and associates performed translumbar and modified transarterial embolization in 84 patients. Endoleak repair was technically successful in 72% treated with the translumbar technique and in 78% treated with the transarterial technique (9). In the present group, clinical success was observed in all patients despite the fact that technical success was not achieved in two patients (ie, filling of endoleak cavity persisted on follow-up CT). In both patients, the endoleak persisted (although it was qualitatively smaller) with no further growth in the aneurysm. Technical failure occurred in cases in which the endoleak cavity could not be successfully catheterized, and embolic materials were instead deposited in lumbar arteries perfusing the endoleak. Nevala and associates (8) reported treatment of type II endoleaks in 14 patients with stable or growing aneurysms. Endoleaks were eradicated in eight of 12 (two patients were lost to follow-up). Interestingly, they concluded that their results of secondary interventions on type II endoleaks were “quite poor” despite the fact that there were no ruptures and all patients who were able to be followed had stable aneurysms after completion of therapy (8). Although it is impossible to draw firm conclusions based on the small numbers of patients in both studies, it appears that decreasing perfusion to the sac may stabilize some growing aneurysms in patients with type II endoleaks, and inability to completely eradicate a small endoleak does not necessarily equate to clinical failure. It merits further study.

In the present cohort, CT scans of variable quality obtained at the study institution and elsewhere appeared to be very accurate for the detection of endoleaks but less accurate in characterizing the endoleak. Nine of 25 patients referred for endoleak management had type I or type III endoleaks that were incorrectly classified as type II endoleaks. Misclassification does not appear to be unique to our hospital, as the present series includes patients referred from more than eight hospitals in three states. Misclassification of type II endoleaks may be attributed to several factors. First, type II endoleaks are far more common than other types; radiologists interpreting these studies and vascular specialists treating these patients have far more experience with this type of endoleak and may be predisposed to label any endoleak as a type II endoleak. This is particularly true because the findings of different types of endoleaks may be very similar on CT; collateral arteries (ie, ascending lumbar artery and IMA) in patients with type I or III endoleaks are often opacified, identical to type II endoleaks because these vessels serve as routes of egress from the aneurysm sac. Second, some type II endoleaks may be indistinguishable from very small type III endoleaks, even on angiography. In five patients, we did not correctly diagnose a type III endoleak until we had performed embolization and saw continued perfusion of the aneurysm sac (Fig 2). In these cases, correct classification could not be made on initial CT, even in retrospect. Although larger type III endoleaks are typically conspicuous on CT and angiography, smaller endoleaks are more difficult to correctly classify. A pinpoint leak of contrast medium into a large perfused cavity within the thrombus of an aneurysm sac may be occult on arterial-weighted CT because contrast medium entering the aneurysm sac through the rent in the graft is diluted by unopacified blood within the cavity. On venous-weighted CT, the cavity within the sac, as well as arteries providing egress, are opacified. As such, a type II endoleak cannot be easily differentiated from a type III endoleak. Because type II endoleaks are far more common, there appears to be a tendency to classify the abnormality as a type II endoleak. When aortography is performed, the pinpoint leak may remain occult as a result of the aforementioned dilutional effect. With selective angiography, the endoleak

may be opacified “retrogradely” via the IMA or lumbar arteries with a forceful injection of contrast medium. This is especially true for complex leaks with several routes of egress. Clearly, a forceful injection at the origin of the IMA can reflux contrast medium into the aneurysm sac and out of lumbar arteries, even in the presence of a small type III endoleak. Thus, before embolization, the abnormality is misclassified as a type II rather than a type III endoleak on both imaging modalities.

Newer techniques show promise for characterization of endoleaks. Dynamic CT image acquisition, referred to as four-dimensional dynamic volumetric CT angiography, consists of using newest-generation scanners to image a volume of tissue continuously over a fixed period of time after intravenous administration of contrast medium. A true angiographic study is obtained, demonstrating the nidus of an endoleak within the aortic sac and how the endoleak is perfused. Bent and associates (12) reported the use of this technique to evaluate a patient with an endoleak of unclear etiology demonstrated on conventional CT angiography after fenestrated endovascular aortic repair. By using the four-dimensional technique, images were acquired continuously in a 16-cm field of view and temporally reconstructed at a rate of two frames per second, enabling precise localization of a type III endoleak.

We have observed that some type III endoleaks may be amenable to endovascular treatment when ethylene vinyl copolymer is used. Although this has not been previously reported to our knowledge, relining the graft to cover the defect seems preferable to embolization for this type of endoleak given the unknown risk of nontarget embolization to the pelvis or legs through the rent. In the present series, two patients with type III endoleaks that were initially misclassified were successfully treated by completely filling the endoleak cavity. This is an important observation because perceived improved efficacy of endoleak treatment with a translumbar approach with liquid embolic agents may reflect the fact that some patients with unrecognized type III endoleaks may be successfully treated with this technique. The misclassification of endoleaks would also help to explain why some leaks appear refractory to embolization of “feeding arteries” with the use of other embolic materials such as coils. Clearly, a patient with a type III endoleak cannot be successfully treated by occluding dominant egress vessels if adjacent arteries remain patent. For example, embolization of patent lumbar arteries would only serve to divert flow into different lumbar arteries. This is not to imply that all patients with growing aneurysms have misclassified endoleaks, but underscores the fact that a type III endoleak should be included in the differential diagnosis of any patient with a growing aneurysm irrespective of CT appearance.

The limitations of the present investigation include that it is a single-institution retrospective study of a small and diverse cohort. Reflecting the gamut of community practice, many patients were referred from outside hospitals by a variety of physicians with nonuniform imaging evaluation. It is clear that the findings of this study have been influenced by variable diagnostic workup, and the value of high-quality imaging and radiologic interpretation cannot be underestimated. Embolization was performed with a variety of embolic agents from variable approaches. Other limitations include the inability to determine how many of our patients underwent anticoagulant therapy at some point during their follow-up period, which may impact presence and persistence of type II endoleaks, and the lack of a control group. Although we believe it was unlikely, we cannot discount the possibility that some aneurysms proven to have increased in size may have subsequently stabilized without any therapy. Finally, we excluded one patient who had been treated at several outside hospitals with a number of different embolic agents. We thought that including this patient would introduce confounding variables because the treatment algorithm used in her care differed from that of the group as a whole.

In summary, two conclusions can be drawn from the present series of patients with enlarging aneurysms attributed to type II endoleaks. First, embolization of type II endoleaks effectively arrests growth; sacs stabilized in all patients with type II endoleaks after embolotherapy. Second, unrecognized type III endoleaks appear to be relatively common in this group of patients. More than one fourth of patients referred for treatment had type III endoleaks rather than type II endoleaks as initially diagnosed on CT.

ABBREVIATION

IMA inferior mesenteric artery

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Figure 1.

Staged algorithm of evaluation and treatment of suspected type II endoleaks. (Available in color online at www.jvir.org.)

Figure 2.

Type III endoleak correctly classified after embolization. **(a)** Arterial-phase enhanced CT shows no evidence of endoleak. **(b)** Venous-phase enhanced CT shows large endoleak cavity (large arrow) and patent IMA and lumbar arteries (small arrow). **(c)** Angiogram obtained via the middle colic artery shows retrograde perfusion of the endoleak cavity. **(d)** Fluoroscopic image shows coil embolization of the IMA origin. **(e)** Enhanced CT after further embolization of lumbar arteries and cavity (not shown) shows coils in the IMA origin (arrow). **(f)** Enhanced CT caudal to **e** shows coils in the ascending lumbar arteries (arrows). **(g)** Enhanced CT cephalad to **e** shows type III leak (arrowheads) from the anterior aspect of the endograft.

Table
Summary of Patients with Type II Endoleak and Proven Aneurysm Enlargement after EVAR

Case	Endograft Type	Endograft Age (mo)	Sac Diameter (cm)	Type of Endoleak	Embolization Technique(s)	Outcome	Follow-up (mo)
1	Excluder	14	6.3	II	TALC × 2	Stable	18
2	AneuRx	75	5.8	II	TALC	Stable	67
3	Excluder	41	6.4	II	TAIC, TALC	Stable	47
4	AneuRx	Unknown	6.2	II	TAISC, DPG	Stable	8
5	AneuRx	Unknown	7	II	TAISC	Stable	12
6	AneuRx	72	6	II	TAL, AR, SC	Decreased by 2.1 cm	18
7	AneuRx	59	8.3	I	None	Open repair	–
8	Zenith	82	5.7	IIIB	TAISC, TALC	Repeat stent-grafting	–
9	Excluder	58	7.6	I	None	Repeat stent-grafting	–
10	Zenith	12	8.5	II	TAISC	Decreased by 3.5 cm	88
11	AneuRx	17	8.9	II	TAIC, DPG	Stable	12
12	AneuRx	66	5.9	IIIB	DPG	Repeat stent-grafting	–
13	Ancure	27	5.7	II	TAISC	Stable	12
14	Ancure	46	8.6	IIIB	TAISC	Repeat stent-grafting	–
15	AneuRx	41	6.2	IIIA	None	Repeat stent-grafting	–
16	Zenith	17	6.6	II	TAISC	Stable	72
17	Excluder	17	6.1	II	TAIC, TALC, DPO	Stable	6
18	Ancure	69	5.5	II	TALC, TALC, DPG	Stable	11
19	Excluder	Unknown	5.8	II	TALC, DPO	Stable	13
20	Excluder	Unknown	9.4	IIIB	TAIC, DPO	Stable	12
21	Excluder	12	5.9	II	TAIC, TALC	Stable	12
22	Excluder	12	5.7	IIIA	None	Repeat stent-grafting	–
23	Excluder	12	7.4	II	TAIC, DPO	Stable	7
24	Zenith	59	6.8	IIIB	TALC, DPO	Stable	9
25	Excluder	46	7.2	II	TAIC, DPO	Stable	6

Note.—AR = accessory renal embolization, DPG = direct puncture cyanoacrylate embolization, DPO = direct puncture ethylene copolymer embolization, EVAR = endovascular aneurysm repair, TAIC = transarterial inferior mesenteric artery coil embolization, TAISC = transarterial inferior mesenteric artery and sac coil embolization, TAL = transarterial lumbar embolization, TALC = transarterial lumbar artery coil embolization, SC = sac embolization.