Initial successful management of type I endoleak after endovascular aortic aneurysm repair with n-butyl cyanoacrylate adhesive

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Objective: **Transcatheter embolization with coils and other agents has been described as a treatment method for type II endoleak after endovascular aortic aneurysm repair (EVAR). Type I endoleak has not been treated commonly with such therapies, although most investigators believe they warrant definitive intervention. The liquid adhesive n-butyl 2-cyanoacrylate (n-BCA) is often used to treat congenital arteriovenous malformations. The objective of this study is to report our initial experience in treating type I endoleak with n-BCA and with a variety of other interventions.**

Methods: **A retrospective review was performed of 270 patients who underwent EVAR at our institution between January 1994 and December 2002. Of these, 24 patients had type I endoleak (8.9%), diagnosed either intraoperatively (n = 13, 52%) or during follow-up (n = 12, 48%). Among these 24 patients, 17 had proximal leaks and the remaining 8 patients had distal leaks. These cases form the focus of this study.**

Results: **Twenty-two leaks required endovascular intervention, with the following success rate: n-BCA, 12 of 13 cases (92.3%); extender cuffs, 4 of 5 cases (80%); coils with or without thrombin, 3 of 4 cases (75%). In one patient with persistent endoleak despite attempted endovascular intervention the device ultimately was surgically explanted, and the patient did well. Of six patients with endoleak initially managed expectantly, two eventually underwent attempts at definitive intervention, both with n-BCA. Three sealed spontaneously before definitive intervention could be performed; and in one 97-year-old patient who refused intervention, the aneurysm subsequently ruptured and the patient died. In total, 13 patients with type I endoleak underwent n-BCA transcatheter embolotherapy. No serious complications were directly related to this therapy. Colon ischemia developed in one patient, and was believed to be a result of thromboembolism during wire and catheter manipulation rather than n-BCA treatment. Twelve of these 13 leaks remain sealed at mean follow-up of 5.9 months (range, 0-19 months).**

Conclusion: **Our initial use of n-BCA occlusion suggests that it may be an effective and safe method of treatment of type I endoleak after EVAR. In particular, n-BCA embolotherapy may be especially useful in treating type I endoleak not amenable to placement of extender cuffs. Larger case series and longer follow-up are needed before this treatment is more broadly recommended. Type I endoleak after EVAR can be treated successfully with a variety of endovascular methods, and surgical explantation is rarely required. (J Vasc Surg 2003;38:664-70.)**

Successful endovascular abdominal aneurysm repair (EVAR) can be defined as complete exclusion of blood flow from the aneurysm sac. Complications of EVAR vary, and include distal graft migration, hematoma, graft limb thrombosis, peripheral embolization, and, most commonly, perigraft leak, otherwise known as endoleak. Endoleak is defined as any blood flow outside the endovascular graft and within the intact aneurysm sac.[1] While type II (branch vessel) leak is debatably benign, type I (attachment site) leak is generally considered to warrant some form of intervention, because of the belief that it represents risk for future rupture.[2-4] The reported incidence of endoleak ranges from 8% to 44%; however, the natural history of this complication remains to be defined and awaits long-term follow-up.[5] Our own experience, as well as the literature, seems to indicate that many branch vessel and even some attachment site endoleaks are capable of sealing spontaneously.[6-8]

Endovascular intervention for type I endoleak has consisted primarily of using an extender cuff or short covered stent to overlap the attachment site leak. More recently, attempts at coil embolization have also been successful.[9-11] Use of liquid adhesives or "glues" has also been described as a possible treatment method for both type I and type II endoleaks.[12] The liquid adhesive n-butyl 2-cyanoacrylate (n-BCA), commonly used to treat congenital arteriovenous malformations, was used by Yamaguchi et al[13] to successfully seal an attachment site leak in a patient after EVAR of the aortic arch.

We report our experience in treating type I endoleak with n-BCA and a variety of other interventions.

METHODS

A retrospective review was performed of a prospectively compiled database of all endovascular abdominal aortic

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aneurysm (AAA) repairs performed at New York University Medical Center. From 1994 through December, 2002, 270 endovascular AAA repairs were attempted. The first 46 (17%) of these were performed during approved clinical trials, including the Endovascular Technologies trial (EVT; Endovascular Technologies, Menlo Park, Calif) and the Excluder trial (W. L. Gore & Associates, Flagstaff, Ariz). The remaining 224 (83%) were performed after approval of the devices by the US Food and Drug Administration (FDA) in 1999. Devices used after FDA approval included the Ancure (Guidant, Menlo Park, Calif), AneuRx (Medtronic, Minneapolis, Minn), and Excluder stent grafts. We routinely oversize endografts by 10% to 15%.

During clinical trials patients underwent follow-up imaging studies according to protocol.[8] Since conducting clinical trials, it has been our institutional practice to obtain a contrast medium–enhanced and non-contrast-enhanced computed tomography (CT) scan within 1 month of repair. Repeat studies are performed at 6 and 12 months, and yearly thereafter. Type I endoleak was defined as originating from either the proximal or distal attachment sites. Greatest minor axis cross-section diameter of the aneurysm sac was measured for each study. Any increase or decrease in aneurysm diameter greater than 0.5 cm compared with previous imaging studies was considered significant.

General indications for arteriography and possible treatment of endoleak included presence of any attachment site leak; any increase in aneurysm size, with or without CT demonstration of endoleak; persistence of endoleak for more than 6 months in aneurysms larger than 6 cm in diameter; and surgeon preference.

Of the 270 patients, 24 patients had type I (attachment site) endoleak demonstrated either intraoperatively or on subsequent imaging studies. This group of patients comprised the focus of our study. Type I leak was considered a primary leak if it was diagnosed intraoperatively or in the perioperative period (<30 days). Any leak diagnosed on a subsequent study (>30 days postoperatively) was considered a secondary leak.

The various intervention methods used were placement of an AneuRx extender cuff or Wallgraft endoprosthesis (Boston Scientific, Natick, Mass); coil embolization with or without thrombin injection; and Trufill n-BCA (Cordis, Miami Lakes, Fla), with or without coil embolization of outflow vessels. An AneuRx extender cuff or Wallgraft was placed when possible as first-line treatment. In patients with anatomy prohibitive for placement of a stent or cuff, embolotherapy was used. Choice of embolic agent was made according to surgeon preference. All interventions were performed by surgeons or interventional radiologists, in the operating room or the interventional suite. For transcatheter embolotherapy, access to the endoleak was obtained transfemorally and retrograde via the graft. Superselective catheters were introduced into the leak and placed in the aneurysm sac (Fig 1).

Inasmuch as n-BCA is FDA-approved solely for use in central nervous system arteriovenous malformation embolization, informed consent was obtained from each patient before off-label use. n-BCA liquid adhesive was prepared by addition of ethiodized oily contrast medium and tantalum powder (optional). These modifications provide n-BCA with radiopacity and slow polymerization time, enabling more precise delivery of the embolic agent. Small volumes (0.2-2.0 mL) are used in each deposition. Scrupulous attention is afforded to maintaining a nonionic environment, thereby preventing premature polymerization and hardening.[14]

For patients who received n-BCA, subsequent noncontrast-enhanced CT scans were compared with contrast-enhanced CT scans to help differentiate calcium or contrast-enriched "glue" from bona fide endoleak. Furthermore, before completing the retrospective review, a radiologist independently reviewed, in blinded fashion, films in which endoleak was either present or absent and was able to consistently identify true leaks (confirmed at angiography) from enhancing artifact in the n-BCA.

RESULTS

Endovascular repair was performed in 270 patients (243 men, 90%; 27 women, 10%). Ancure grafts were placed in 238 patients (88.1%), Excluder grafts in 15 patients (5.6%), and AneuRx grafts in 17 patients (6.3%). Fourteen endografts (5.2%) were tube configured, and 256 (94.8%) were bifurcated devices.

Type I endoleak detection and presentation. A summary of type I leak presentation is presented in Table I. Twenty-five type I leaks were detected in 24 of 270 patients (8.9% [20 men, 83%; 4 women, 16%). Devices implanted in these 24 patients included 20 bifurcated EVT/Ancure grafts, 2 tube EVT/Ancure grafts, and 2 AneuRx grafts. The two tube endografts were among the earlier devices implanted; one of these was implanted in a patient who had undergone previous abdominal aneurysm repair, with subsequent development of a proximal anastomotic pseudoaneurysm.

Of the 25 type I endoleaks, 17 were proximal (68%) and 8 were distal (32%). Eighteen endoleaks (71%) were primary; 13 (52%) were detected intraoperatively, and 5 (20%) were detected perioperatively, on the first follow-up CT scan at 1 month). Seven endoleaks (29%) were considered secondary, diagnosed at routine follow-up imaging more than 1 month postoperatively and subsequent to a normal CT scan. All leaks were asymptomatic when diagnosed.

Treatment. A summary of all management and treatment strategies is presented in Table II. Success is defined as resolution of the endoleak and stabilization or shrinkage of the aneurysm sac. Of the 25 type I leaks, 19 were treated with endovascular interventions and 6 were initially managed expectantly. Interventions were considered primary if they were the first intervention performed to treat a leak, and secondary if they were an intervention intended to treat a leak that persisted after primary intervention or expectant management. Three secondary interventions were performed, two with n-BCA to treat unsuccessful expectant management and one with an extender cuff and eventually

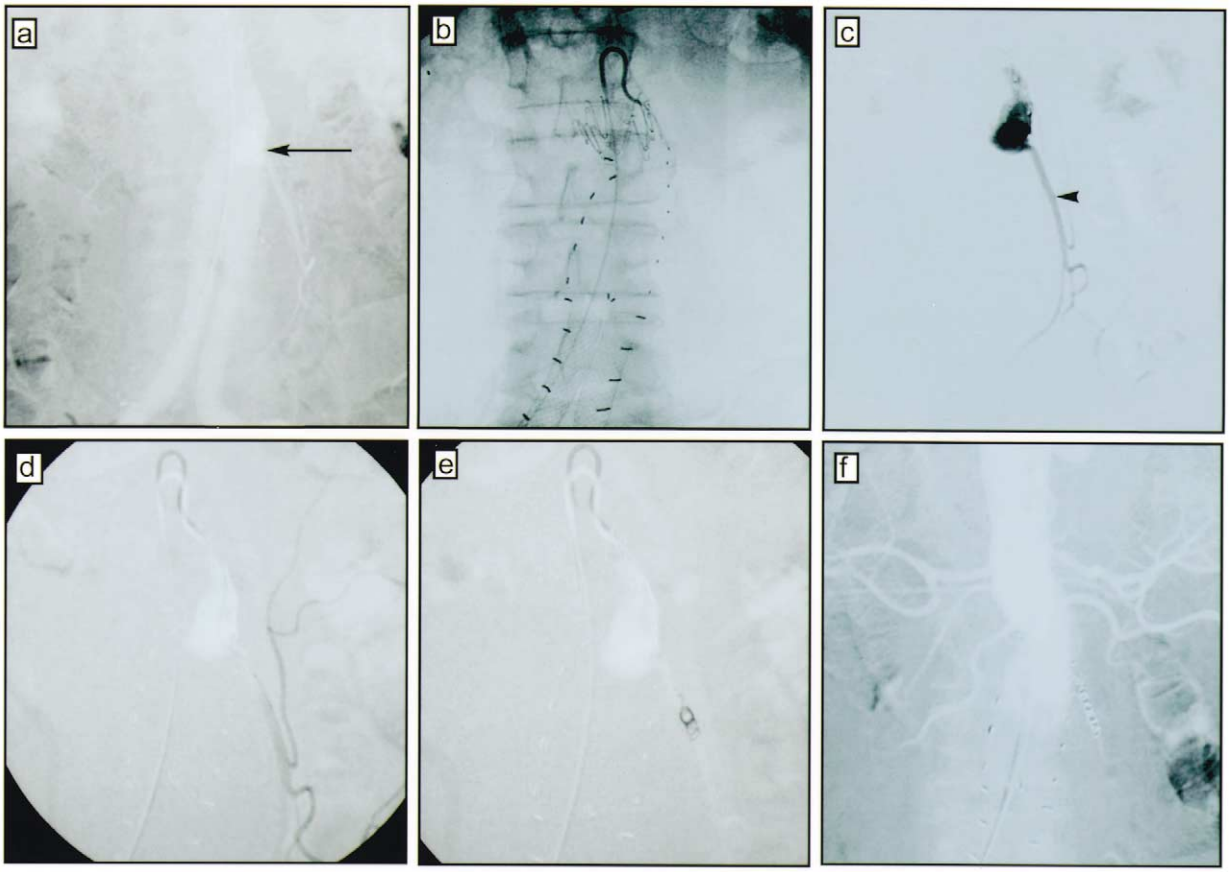

Fig 1. A, Aortogram demonstrates large proximal type I endoleak *(arrow)*. **B and C,** Superselective catheter is used to probe the proximal cuff and access the aneurysm sac, whereupon a selective angiogram is obtained. Note inferior mesenteric artery (*arrowhead* in **C**) serves as an outflow vessel in this "mixed" leak. **D,** Inferior mesenteric artery is coil-embolized to prevent subsequent nontarget embolization of n-BCA. **E,** n-BCA, 0.2 to 2 mL, is used to embolize the proximal type I endoleak. **F,** Completion aortogram demonstrates absence of previous type I endoleak.

Table I. Presentation of type 1 endoleak

Site	*Primary*				*Secondary ("delayed")*			
	Intra-operative		*<1 Mo*		*1–6 Mo*		*>6 Mo*	
	n	*%*	*n*	*%*	*n*	*%*	*n*	*%*
Proximal (n = 17)	9	52.9	3	17.6	1	5.9	4	23.5
Distal (n = 8)	4	50	2	25	1	12.5	1	12.5
Total (n = 25)	13	52	5	20	2	8	5	20

Table II. Summary of all interventions for type I endoleak

Intervention	*Coil*	*n-BCA*	*Extender cuff*	*Expectant*	*Explantation*
First	4 (1)	11 (1)	4	6 (3)	0
Second	0	2	1 (1)	0	0
Third	0	0	0	0	1
Total	4	13	5	6	1

Numbers in parentheses represent treatment failures.
n-BCA, n-Butyl cyanoacrylate adhesive.

open repair to correct a persistent proximal leak initially treated with coil embolization. A summary of the success rates for each treatment strategy is presented in Fig 2.

n-BCA. Of 25 type I leaks, 13 (52%) were treated with n-BCA. Eleven (44%) were primary interventions, and two (8%) were secondary interventions to treat leaks that had not sealed after more than 6 months despite expectant management (Table III). Ten of 13 leaks (77%) treated with n-BCA were proximal, and 3 (23%) were distal. When identified (n = 5, 38%), outflow vessels were coil-embolized before casting the sac with n-BCA and sealing the inflow to the leak. All 13 leaks treated with n-BCA were considered successfully sealed at angiography at completion of the intervention. One of the three distal attachment site leaks sealed with n-BCA showed evidence of contrast medium in the iliac aneurysm sac on follow-up images at 6

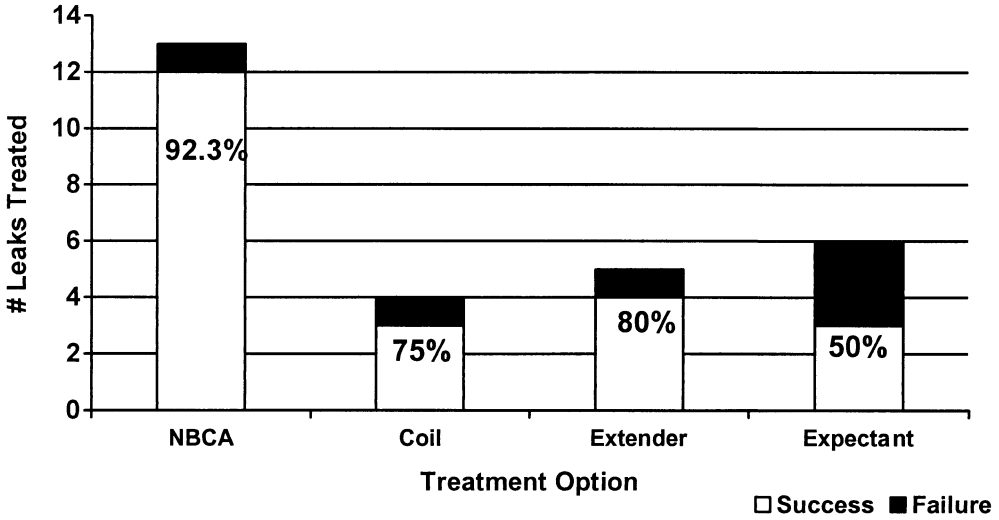

Fig 2. Management of type I endoleak.

Table III. n-BCA embolization of type 1 endoleak

Timing of n-BCA embolization	*No. of leaks treated with n-BCA*
Primary, intraoperative	4
Primary, <1 mo postoperative	2
Secondary, >1 mo postoperative	5
After unsuccessful expectant management	2
Total	13

Primary endoleaks include any leak diagnosed intraoperatively or on first imaging study (<1 month postoperatively). Secondary endoleaks are those detected >1 month postoperatively or after a previous normal imaging study.
Note: n-BCA was used to treat two leaks initially managed expectantly but that persisted at 6 and 10 months, respectively.
n-BCA, n-Butyl cyanoacrylate adhesive.

months and represented the only failure of n-BCA to date. Of note, the intravenous contrast medium was contained within the iliac aneurysm in this patient, suggesting that the "glue" or fabric seal was intact more proximally and the aortic aneurysm sac remained excluded from the circulation. Mean follow-up for leaks treated with n-BCA was 5.9 months (range, 0-19 months).

Coil embolization. Four (3 proximal, 1 distal) of 25 type I leaks (16%) were treated solely with coil embolization. One of the three (33%) proximal type I leaks coil-embolized persisted and required a secondary intervention. Initial attempt at placement of an extender cuff failed, and conversion to open repair was required. The remaining three type I endoleaks treated with coil-embolization were considered successfully sealed at angiography at completion of the intervention. Mean follow-up for leaks treated with coil embolization was 25 months (range, 17-40 months).

Extender cuffs. Of 25 type I leaks, 5 (20%) were treated with AneuRx extender cuffs or covered Wallgrafts. Four (16%) were primary interventions, and one (4%) was a secondary intervention to treat a leak that persisted despite attempts at coil embolization at initial EVAR 2 months previously. This attempt at covering the proximal type I leak with an extender cuff was unsuccessful, and subsequent open repair was required. A previously underappreciated thoracoabdominal aneurysm was discovered at open repair. Follow-up for leaks treated with extender cuffs was 9.3 months (range, 1-24 months).

Expectant management. Of 25 type I leaks, 6 (24%) were initially managed expectantly, 5 proximal (83%) and 1 at the distal attachment site (17%). Four of these leaks were diagnosed intraoperatively. The one distal type I leak was diagnosed on the first follow-up CT scan at 1 month. Reasons for no initial intervention included surgeon preference (n = 4) and patient refusal of further intervention (n = 2). Two (33%) of the six leaks initially treated expectantly demonstrated increasing aneurysm sac diameter on follow-up imaging studies, and n-BCA embolization was successfully performed at 6 months and 10 months, respectively. Three (50%) of the six leaks managed expectantly sealed spontaneously. The final patient (16%) of the six whose leaks were managed without intervention refused treatment at 1 month; the aneurysm subsequently ruptured, and the patient died 6 months post-EVAR, presumably as a result of the untreated distal attachment site endoleak. Follow-up in the three patients in whom expectant treatment was successful was 13.6 months (range, 12-17 months).

Complications related to intervention. No major or minor complications were directly related to any of the various interventions among the 25 type I endoleaks. In one patient, colon ischemia developed after EVAR. Although nontarget embolization cannot be definitively ruled out as a potential cause of this complication, there was

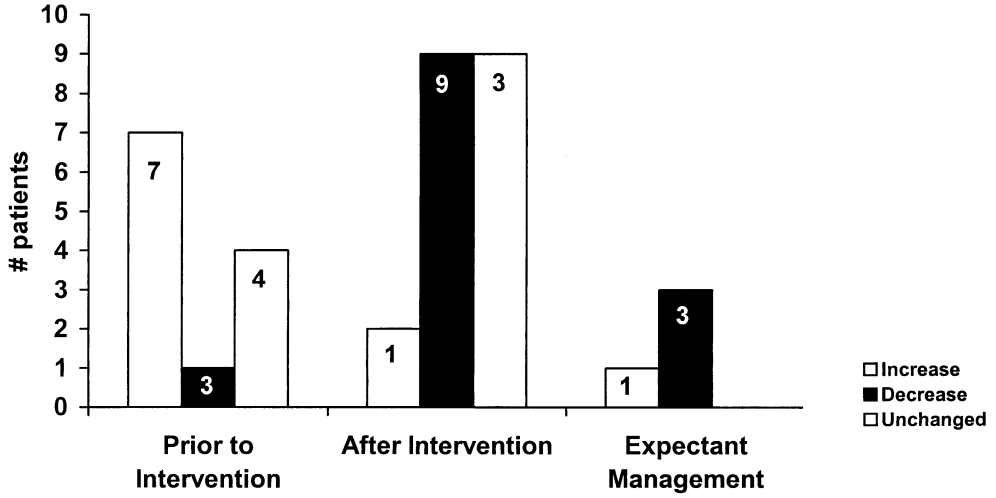

Fig 3. Trends in aneurysm maximal diameter in patients with type I endoleak.

a substantial amount of thrombus in the aneurysm, and we believe the complication to be a result of thromboembolism during wire and catheter manipulation, rather than n-BCA treatment itself. Furthermore, because of the radiopacity of n-BCA, the glue was easily visualized throughout the procedure, and nontarget embolization should have been appreciated, had it occurred.

Morbidity and mortality. Four of the 24 patients with type I endoleak died during follow-up. One 97-year-old patient died within 1 year of surgery, of unrelated causes, with no evidence of AAA expansion or rupture; one patient died of metastatic disease from colon cancer diagnosed post-EVAR; one patient died of sepsis after a prolonged postoperative course after EVAR, complicated by colonic ischemia; and one patient died after rupture of the abdominal aneurysm 6 months post-EVAR. In this last patient, a large distal attachment site leak was diagnosed at CT performed 1 month postoperatively, after implantation of an EVT tube graft. The patient refused intervention, and 6 months later came to the emergency room hemodynamically stable but with increasing abdominal pain. The diagnosis of contained rupture was confirmed at CT. The patient again refused intervention, and died.

Perioperative morbidity in the 24 patients with type I endoleak included severe buttock ischemia and compartment syndrome, with transient renal failure, in one patient, and, in another patient, an expanding groin hematoma that developed on the first postoperative day and required evacuation in the operating room.

Aneurysm sac maximal diameter measurements. Of the 24 patients with type I endoleak, trends in aneurysm maximal diameter were examined before intervention, and after intervention when appropriate. A summary of trends in aneurysm sac maximal diameter is presented in Fig 3. Sac measurement before intervention was possible only in the 12 patients for whom interim imaging studies were obtained. Among these 12 aneurysm sacs, 7 increased in size, 1 decreased in size, and 4 remained unchanged. Sac measurements made after intervention were possible in 21 of 24 patients; 3 patients are awaiting scheduled follow-up studies. Among these 21 aneurysm sacs, 3 increased in size, 9 decreased in size, and 9 remained unchanged. Of the three sacs that increased in maximal diameter, one was in the patient with rupture after refusing intervention to treat a distal type I leak; one was in a patient with a large type II endoleak that became evident 12 months after n-BCA embolization of a distal attachment site leak; and one was in a patient with a distal type I endoleak that persisted despite attempts at n-BCA embolization. This last patient awaits further intervention.

DISCUSSION

Type I endoleak is generally considered to warrant some form of intervention, because of the belief that it represents risk for future rupture.[2-4] A recent literature review by Bernhard et al[15] found 47 reported ruptures secondary to endoleak after EVAR to date, with overall mortality of 50% and operative mortality of 41%. Most of these leaks originated at the attachment site.

A common approach to treating type I endoleak involves additional balloon dilation. Alternatively, covering the attachment site leak with an extender cuff or Palmaz stent may be attempted. However, this is an option only if sufficient native aorta is available proximal or distally to support the stent. An endograft abutting significant arterial branches precludes use of an extender cuff or covered stent. In this setting coil embolization has emerged as an attractive alternative, with good results.[9,16] Nevertheless, the potential for recanalization and continued transmission of systemic pressure through the thrombus and surrounding coils are concerning possibilities.

Casting the attachment site leak with "glue" is an additional treatment option that has been reported only anecdotally.[13] Routinely used for treatment of cerebral arteriovenous malformations, glues such as n-BCA require some expertise if used intravascularly.[17,18] A serious potential complication of embolotherapy is ischemic injury from nontarget embolization. This is more likely to occur if the solution is not viscous enough. To avoid this, we attempted to coil-embolize outflow vessels when present, before injecting n-BCA in the sac or the leak. A further pitfall regarding use of n-BCA is the possibility of premature polymerization or delayed withdrawal of the delivery catheter, either of which can result in gluing the catheter tip in place.

We first attempted n-BCA embolization for treatment of type I endoleak in patients with unfavorable anatomy (eg, short neck) that prohibited safe placement of extender cuffs to seal a proximal attachment site endoleak. These patients were ideal candidates for coil or glue embolization. When possible, we prefer n-BCA to coil embolotherapy, which can be laborious and time-consuming and often requires placement of multiple coils. Furthermore, in theory, coil embolization may be prone to recanalization, because coils act by forming a nidus for thrombus. n-BCA, when used to treat arteriovenous malformations, is resistant to recanalization and may be more durable.[14] Finally, coil embolization has the disadvantage of producing significant artifact, which may confound follow-up imaging studies. Although contrast-impregnated n-BCA may pose a problem in differentiating a new endoleak from glue artifact, this can be resolved by comparing CT images obtained with and without intravenous contrast medium.

In our study, 24 of 270 patients (8.9%) undergoing EVAR had a total of 25 type I endoleaks. While some authors discount reporting an endoleak detected on an intraoperative study but that has resolved by the subsequent follow-up, we included all leaks, regardless of time of presentation.[19] Moreover, we were reluctant to have the patient leave the operating room with a type I endoleak, and chose to intervene in most cases. Overall, 19 leaks were managed with initial intervention: 11 with n-BCA, 4 with extender cuffs, and 4 with coils or thrombin. Of 6 leaks managed expectantly at the initial setting, 2 eventually underwent attempts at definitive intervention, both with n-BCA. Of the 22 endoleaks in which endovascular intervention was attempted, each treatment method fared comparably well. Success rates were as follows: n-BCA, 12 of 13 cases (92.3%); extender cuffs, 4 of 5 cases (80%); and coils with or without thrombin, 3 of 4 cases (75%). One distal type I endoleak treated with n-BCA persisted, and was considered the only treatment failure among the leaks treated with "glue." In truth, the aortic aneurysm sac remained excluded from the systemic circulation with the n-BCA; however, because an aneurysm developed in the common iliac artery over time, intravenous contrast medium pooled around a free-floating limb and, by definition, was categorized as a distal attachment site endoleak. Such rigorous definition may be excessive when exclusion of the aortic aneurysm sac is not compromised. Alternatively, n-BCA may not be advisable for treatment of distal type I endoleak if the iliac system is aneurismal, especially if an extender cuff would suffice. Indeed, n-BCA may be best suited for treatment of a proximal attachment site leak in the setting of a short neck that would not accommodate an extender cuff or Wallgraft without impinging on the renal arteries.

Six of 25 type I endoleaks (24%) were managed expectantly. Three of the six (50%) sealed spontaneously, two persisted and necessitated secondary intervention with n-BCA embolization, and in one patient rupture occurred after refusal of treatment of a known distal type I endoleak. While spontaneous resolution of endoleaks is well- described, it may be only temporary.[8] Mialhe et al[6] showed that 20% of type I leaks that spontaneously seal reopen at 12 to 18 months. Thus lifelong careful vigilance is essential.

Seven endoleaks (29%) were considered "delayed" or secondary, diagnosed at routine follow-up imaging more than 1 month postoperatively and subsequent to a normal CT scan. Although delayed endoleak occurs, the natural history has yet to be defined. Schurink et al[20] reviewed 23 publications with a combined total of 1118 patients and found that 27% of detected leaks were considered delayed. Whether these delayed endoleaks were truly new or were simply missed is debatable.

Attachment site endoleak resulting from device migration is a well-recognized phenomenon that may occur more often after EVAR when a device without a hook attachment system is used. Of note, in most of our patients the hook-based Ancure endograft was implanted. Only two of our patients with type I endoleak received an AneuRx device, which relies on friction and radial force, rather than hooks, for attachment. Both of these endoleaks (one intraoperative, one detected at 1 month) were treated with n-BCA; however, neither leak occurred as a result of migration, but most likely resulted because of poor patient selection (short angulated neck). In instances of migration, an extender cuff or stent is indicated, rather than embolotherapy of any kind.

In conclusion, our initial use of n-BCA occlusion suggests that this may be a viable option for treatment of type I endoleak after EVAR. In particular, n-BCA may be ideal for treating endoleaks in patients with anatomy unsuitable for placement of an extender cuff. Among issues that remain to be elucidated are the effect of n-BCA adhesive on endotension, its biocompatibility with aneurysm sac and graft fabric over time, and the effect of sac remodeling on the glue cast. Larger case series and longer follow-up are needed before this treatment is more broadly recommended. Until then, type I endoleak after EVAR can be treated successfully with a variety of endovascular methods, and surgical explantation is rarely required.

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