Technique and results of transfemoral superselective coil embolization of type II lumbar endoleak

Karthikeshwar Kasirajan, MD, Brian Matteson, MD, John M. Marek, MD, and Mark Langsfeld, MD, Albuquerque, NM

Objective: This study was undertaken to describe the technique of transfemoral superselective coil embolization of type II endoleak and its influence on abdominal aortic aneurysm diameter.

Methods: Over 23 months, 104 aortic stent grafts were deployed to exclude abdominal aortic aneurysms, at an academic medical center. Increase in aneurysm diameter and perigraft findings on contrast material–enhanced computed tomography scans prompted arteriography. Procedures were performed solely by vascular surgeons in a surgical angiography suite. In 7 patients aneurysm access was via the iliolumbar branches of the internal iliac artery, and in 1 patient aneurysm access was via the inferior mesenteric artery through the arc of Riolan from the superior mesenteric artery. Coaxial catheters were placed to gain access to the aneurysm (8F to 5F to 3F, or 5F to 3F). A 3F Tracker18 was the most distal catheter through which an assortment of 0.018 microcoils were deployed within the aneurysm, and the origin of the feeding vessels when possible.

Results: Aneurysm diameter increased 0.48 ± 0.2 cm over 10.8 ± 5 months before superselective coil embolization. In 6 of 8 patients superselective coil embolization resulted in a mean decrease in aneurysm diameter of 1.3 ± 1.2 cm over 9 ± 3.2 months. Failure was presumed due to inability to reach the aneurysm sac in 1 patient and was associated with oral anticoagulation in 1 other patient.

Conclusion: Proper identification of the source of type II endoleak and its complete occlusion, combined with aneurysm sac coiling, may result in prompt decrease in aneurysm size. (J Vasc Surg 2003;38:61-6.)

Encouraged by favorable early results of aortic endograft to treat infrarenal abdominal aortic aneurysm, this procedure has continued to gain popularity. The primary goal of therapy is to prevent aneurysm rupture. Increase in aneurysm diameter or volume is considered failure of therapy, and often secondary interventions are necessary to address the cause of aneurysm enlargement. Various mechanisms of device failure have been reported, resulting in persistent pressurization of the aneurysm sac. This may be noted at radiography as contrast blush within the aneurysm, either on computed tomography (CT) scans or diagnostic angiograms. The term endoleak was coined to describe this condition. Type II endoleak is the most common form and results from retrograde filling of the aneurysm from the lumbar, inferior mesenteric, accessory renal, or sacral arteries. The significance and natural history of type II endoleak has not been completely defined.

From the Division of Vascular Surgery, University of New Mexico, Albuquerque, NM.

Competition of interest: none.

Presented at the Seventeenth Annual Meeting of the Western Vascular Society, Newport Beach, Calif, Sep 22-25, 2002.

Reprint requests: K. Kasirajan, MD, University of New Mexico School of Medicine, Division of Vascular Surgery, 2-ACC, 915 Camino de Salud NE, Albuquerque, NM 87131-5341. (e-mail: kkasirajan@salud.unm.edu).


0741-5214/2003/$30.00 + 0

doi:10.1016/S0741-5214(03)75467-0

However, reports of aneurysm rupture after persistent type II endoleak has been described. We report our experience with superselective coil embolization performed to treat type II endoleak.

PATIENTS AND METHODS

Over 23 months, 104 aortic stent grafts (AneuRx, Medtronic/AVE, Santa Rosa, Calif; Ancure, Guidant, Indianapolis, Ind) were deployed to exclude abdominal aortic aneurysm, at an academic medical center. No patients were discharged to home with type I endoleak. All patients underwent follow-up CT at 6-month intervals. Eight patients with type II endoleak apparent on 6-month follow-up CT scans underwent aortography. All 8 patients were men, with mean age of 68.9 ± 7 years and mean preinterventional aneurysm diameter of 6.5 ± 1.3 cm. Increased aneurysm diameter (0.48 ± 0.2 cm over 10.8 ± 5 months) was noted on CT scans in 5 of 8 patients, with obvious contrast blush seen along the periphery of the aneurysm sac, suggestive of type II endoleak. All 8 patients were offered angiography to identify and possibly treat the endoleak, because it was our policy to aggressively investigate and treat all type II endoleak with or without change in aneurysm sac diameter. One patient was receiving warfarin sodium anticoagulation therapy because of chronic atrial fibrillation. For initial aneurysm exclusion, an AneuRx endograft was used in 6 patients and an Ancure graft was used in 2 patients. In 2 patients, both with AneuRx grafts, an initial decrease in diameter after endograft placement was followed by an increase in aneurysm diameter, and a new-
onset type II endoleak was seen on CT scans. In 1 of these patients the aneurysm sac had decreased from 4.8 cm to 4 cm over 6 months, then increased to 4.7 over the next 12 months. In the other patient the aneurysm sac had decreased from 6 cm to 4.5 cm, and subsequently increased to 6 cm over 8 months.

**Technique of superselective coil embolization.** Percutaneous femoral access was used in all patients. In 2 patients in whom the stent graft was extended to the external iliac artery the contralateral femoral artery was used for access, to have access to the ipsilateral internal iliac artery should this prove to be the site of origin of the type II endoleak. Initial angiography was performed through a diagnostic pigtail catheter positioned above the endograft at the level of the renal arteries, with 8 mL of contrast material per second, to a total of 35 mL. Prolonged run times were used to enable visualization of any delayed filling of the aneurysm sac. Then additional contrast material was injected, ie, 5 mL/s to a total of 25 mL, with the diagnostic catheters in the iliac limbs. If the vessel feeding the aneurysm was not yet visualized, selective cannulation of the internal iliac artery followed by selective cannulation of the superior mesenteric artery was performed. Bilateral femoral artery access was obtained if necessary, and was required in 4 patients because the initial entry site was the right common femoral artery and the endoleak was noted to arise from the left internal iliac artery. All type II endoleaks from the iliolumbar arteries were branches of the left internal iliac artery, and all entry sites were on the left of the aneurysm at L3-L4. All patients received systemic anti-coagulation with heparin, as is our practice in all superselective catheterization techniques.

In 7 patients the aneurysm sac was found to fill from branches of the left internal iliac artery. An ipsilateral femoral approach was used in all 7 patients. Selective cannulation of the origin of the internal iliac artery was performed with a 5F right internal mammary artery catheter or Sos catheter (both from Cook Inc, Bloomington, Ind). With a road-mapping technique, selective cannulation of the vessel supplying the iliolumbar arteries, responsible for the type II endoleak, was performed with an angled 0.035 glide wire (Boston Scientific/Target, Natick, Mass) through the right internal mammary artery or Sos catheter positioned at the origin of the internal iliac artery. Subsequently, the catheter was withdrawn and exchanged for an angled glide catheter (Boston Scientific/Target) over the 0.035 glide wire, and this catheter was passed as far up the iliolumbar arteries as technically feasible. Then the glide wire was withdrawn, and a 3F catheter with an 0.018 guide wire (Tracker18 catheter; Boston Scientific/Target) was coaxially passed through the 5F angled glide catheter toward the aneurysm sac. Road-mapping was liberally used to guide catheter placement along the way.

In two patients the vessel supplying the iliolumbar arteries originated from the internal iliac artery, and branched off immediately after the internal iliac artery takeoff. Selective cannulation of this branch was technically impossible because the catheter tended to easily flip out of

the internal iliac artery. Instead of attempting a transbrachial approach, we placed a 7F or 8F Balkan sheath (Cook Inc) at the origin of the internal iliac artery and held it in place with a Rosen wire (Boston Scientific) extending into the distal internal iliac artery (Fig 1, A). In both patients the Balkan sheath was introduced from the ipsilateral femoral access site (Fig 1, A-C). The glide catheter and the Tracker18 were subsequently passed along the side of the Rosen wire through the Balkan sheath (Fig 1, A-C). A 7F or 8F Balkan sheath was required because the Rosen wire was left in place and the 5F angled glide catheter was inserted alongside this wire (Fig 1, B); a smaller caliber Balkan sheath would have required withdrawal of the Rosen wire, thereby losing the additional support in the internal iliac artery that was provided by this “buddy wire” technique. Once access to the aneurysm sac was gained, the 3F catheter was manipulated through the aneurysm to cannulate the outflow branch. An assortment of microcoils were used to embolize the outflow vessel, aneurysm sac, and inflow vessels. For the sake of description, the artery accessed to cannulate the aneurysm sac is referred to as “inflow,” and arterial branches noted with injection of contrast medium into the aneurysm sac are referred to as “outflow.” An attempt was always made to coil the outflow vessels and aneurysm sac (Fig 2, A and B).

In 2 patients we were unable to reach the aneurysm sac because of a large number of extremely small and tortuous source vessels. A liquid embolic agent (D-Stat; Vascular Solutions Inc, Minneapolis, Minn) was used to thrombose these multiple feeding vessels, in addition to coil embolization (Fig 3, A and B). D-Stat, a mixture of thrombin and collagen, was used because it was readily available in the Duett (Vascular Solutions Inc) femoral closure device package. Thrombus formation requires interaction with blood, and hence this had the advantage of not occluding the Tracker18 catheter, which could be left in place for subsequent contrast material injection and additional interventions. Plain thrombin was not used, because it is highly fluid and hence has the propensity to diffuse farther away from the target site.

In 1 patient the aneurysm was fed from the inferior mesenteric artery via the meandering mesenteric artery (Arc of Riolan) from the superior mesenteric artery. Selective transfemoral cannulation of the superior mesenteric artery was performed with a Cobra 2 catheter (Cook Inc), and with road-mapping we were able to coaxially pass the 3F Tracker18 catheter to the aneurysm sac (Fig 4, A). Once inside the aneurysm sac, the outflow lumbar artery was identified and cannulated. Coil embolization was performed on the outflow lumbar artery, aneurysm sac, and inferior mesenteric artery inflow to the aneurysm sac. Care was taken not to place coils in the meandering mesenteric artery, to prevent ischemic complications to the colon (Fig 4, B).

**RESULTS**

The lumbar artery or the inferior mesenteric artery feeding the aneurysm sac was identified in all 8 patients.
Access to the aneurysm sac was achieved in 6 of 8 patients. In the 2 patients in whom we were unable to reach the aneurysm sac (retiform anastomosis) the feeding vessels were coiled and then injected with a mixture of collagen and thrombin (D-Stat). Thrombosis of all feeding vessels was angiographically visualized after injection of the liquid embolic agent; however, in these 2 patients coiling of the actual aneurysm sac was not possible. On the basis of initial angiographic appearance, no filling of the aneurysm sac was noted in any patients at completion of the coiling procedure.

After superselective coil embolization, the aneurysm sac demonstrated reduction of aneurysm diameter in 6 of 8 patients (mean decrease, 1.3 ± 1.2 cm over 9 ± 3.2 months). In 1 patient receiving warfarin sodium anticoagulation therapy the aneurysm continued to fill with a small type II endoleak through the coils deployed in the previous intervention. Warfarin anticoagulation was discontinued, but endoleak persisted at the site of coil embolization. The patient has refused further therapy, because of advanced age and debilitated status. The last CT scan obtained demonstrated a 4 mm increase in aneurysm diameter.
In another patient, in whom we were unable to reach the aneurysm sac, type II endoleak persists despite liquid embolization of all feeding lumbar vessels. No increase in aneurysm diameter was noted at 6-month follow-up, and the patient has opted for CT follow-up at another 6 months. It could not be ascertained whether the persistent type II endoleak was from the same source vessel, because the patient currently refuses further angiographic evaluation.

No major complications have been observed. In the patient receiving warfarin anticoagulation therapy and with persistent type II endoleak, severe buttock claudication developed and had not resolved at 9 months of follow-up. In this patient the contralateral internal iliac artery was embolized and the AneuRx stent limb was extended to the external iliac artery at primary stent graft placement. This was done to exclude a common iliac aneurysm at endograft placement. However, the buttock claudication developed subsequent to coil embolization, on the ipsilateral side, probably indicating that one of the branch vessels that were coiled may have been an important collateral vessel to the gluteal muscles.

**DISCUSSION**

Transarterial coil embolization to treat type II endoleak has been performed with varying degrees of success. Failure was often the result of inability to reach and coil the aneurysm sac. In 5 of our 6 patients in whom direct sac access was possible the endoleak was excluded successfully, with demonstrable decrease in aneurysm diameter. However, transarterial coil embolization is not a simple procedure, and requires advanced catheter skill and knowledge of use of microcatheters. We chose to use coils rather than liquid embolic agents, because of the propensity of liquid embolic agents to diffuse into non-target vessels with catastrophic results. Colonic necrosis as a result of mesenteric thrombosis, and permanent nerve injury have been reported after use of liquid embolic agents. We chose to use liquid embolic agents only when attempts to gain access
to the aneurysm sac failed. In 2 patients the liquid embolic agent was combined with coil embolization to prevent free diffusion of the liquid agent. In 1 of these patients with a small type II endoleak we were able to coil the lumbar arteries leading to and from the aneurysm, and the site of entry into the aneurysm, and in addition we used D-Stat to seal the lumbar arteries. However, type II endoleak persists because direct sac embolization failed. Because this type II leak is small, we did not believe it would be amenable to direct translumbar sac puncture. Currently this patient is being followed up with serial CT. At 6-month follow-up, CT scans did not demonstrate increase in diameter of the aneurysm, despite persistent endoleak.

When an aneurysm continues to enlarge after placement of an endograft, the procedure should be considered failed and further, aggressive investigation should be carried out, with or without evidence of endoleak on CT scans. However, presence of type II endoleak in aneurysms that are not expandable or are even decreasing in size has resulted in varying approaches to treatment. The EUROSTAR experience did not reveal a higher incidence of aneurysm rupture in stable aneurysms with type II endoleak (0.52% rupture incidence) compared with stable aneurysms with no endoleak (0.25% rupture incidence). Although this did not reach statistical significance, the incidence was almost two times greater than with aneurysms with no endoleak, suggesting the possibility of a type II statistical error. Parry et al demonstrated that type II endoleak is associated with a 2 mm increase in aneurysm diameter, compared with a 3 mm reduction in aneurysm diameter in the absence of type II endoleak. Inasmuch as no conclusive evidence indicates otherwise, it has been our practice to offer further interventions to treat all type II endoleak that persists at 6-month follow-up. Because coil embolization is reported to be safe and effective, we offer this as the first treatment option. Although liquid embolic agents are less expensive, complications occur more frequently their use. Various other techniques, e.g., direct CT-guided sac puncture and laproscopic ligation of the lumbar arteries, have been described, but have not gained widespread acceptance. It should also be noted that not all experts in the field are of the opinion that all type II endoleak should be aggressively treated, reserving therapy only in the event of nonshrinking or enlarging sac diameter. In a recent consensus conference, 85% of respondents indicated that transarterial coil embolization of the aneurysm sac, via the hypogastric artery or superior mesenteric artery, is their preferred method for management of type II endoleak.

In our experience, the main disadvantage of performing coil embolization to treat type II endoleak is the artifact that is produced on follow-up CT scans. This artifact often makes it impossible to detect any contrast material extravasation at the site of the previous endoleak. Hence a decrease in the size of the aneurysm sac may be the best, and only, indicator of successful coil embolization of type II lumbar endoleak. Although duplex ultrasound scanning has been used by few authors to follow up and detect endoleak, we have not had much success with this technique. This is similar to the consensus of the international conference: 69% believe duplex ultrasound scanning is not the best method for detection of endoleak.

The high late failure rate reported with transarterial coil embolization of type II endoleak may be explained by failure to coil the aneurysm sac. Baum et al reported a 92% success rate if the sac was directly coiled with translumbar puncture; in contrast, an 80% failure rate was noted with transarterial coil embolization of the feeding vessels. This model of actual coiling of the aneurysm sac was used in 6 of our patients, the only difference being transarterial access to the aneurysm sac. Hence, on the basis of the report by Baum et al and our results, we believe isolated coiling of feeding vessels is predictive of a high failure rate. This was also noted in a recent publication by the group from Southern Illinois University who, despite initial angiographic appearance of success with coil embolization of the feeding vessels, reported a 60% failure rate after subsequent evaluation of CT scans. Despite their attempt to classify type II endoleak on the basis of complexity of the feeding vessels, this had no effect on success of treatment, because direct sac access often was not achieved. On the basis of our results, our treatment algorithm for type II endoleak currently involves an initial attempt at transarterial access to the aneurysm sac; if the aneurysm sac cannot be reached, the patient is referred to a radiologist for CT-guided translumbar sac puncture.

Various authors have demonstrated the influence of the number and size of patent aortic branches arising from the aneurysm and their relationship to occurrence of type II endoleak. Parry et al demonstrated no endoleak in the absence of patent side branches, compared with a 62% endoleak rate with patent lumbar arteries. However, we have not attempted preoperative coil embolization of lumbar or inferior mesenteric vessels. Gould et al reported no decrease in incidence of type II endoleak after preoperative embolization, and concluded that this was an ineffective procedure. The failure of preoperative coiling of branch vessels may have failed because of inability to identify and coil all aortic side branches; this form of complex network of collateral channels has subsequently been clearly demonstrated.

The requirement for direct aneurysmal sac thrombosis with embolic agents may be further substantiated by the work of Walker et al. These authors packed the aneurysm sac with collagen sponge at primary endografting, which significantly reduced the incidence of type II endoleak by achieving complete aneurysm thrombosis, even in patients with multiple aortic side branches. However, Marty et al in experimentally induced aortic aneurysms, failed to show a reduction in pressure within the aneurysm after embolization with metal coils used to treat direct antegrade endoleak. However, no details of the embolization technique or status of feeding vessels were provided.

Although technically challenging, the ability to perform transarterial coil embolization may be readily acquired by vascular surgeons dedicated to becoming multifaceted endovascular specialists. To our knowledge, this is the first
report of superselective coiling of endoleak solely performed by vascular surgeons in a self-contained operating room angiography suite equipped with a ceiling-mounted system and a 15-inch image intensifier. The results may also reflect the superior imaging required for these procedures. Use of portable C arms for complex time-consuming vascular interventions may fail, not because of technical inability but as a result of poor imaging. Although the immediate results of embolization of type II endoleak are promising, long-term results with this technique are not known. Whether long-term reduction in aneurysm sac pressure is achieved with coil embolization remains unknown.

In conclusion, we believe complete embolization of all inflow and outflow vessels, combined with embolization of the aneurysm sac, may provide the most durable treatment of type II endoleak.

REFERENCES