CASE REPORTS

Colonic necrosis subsequent to catheter-directed thrombin embolization of the inferior mesenteric artery via the superior mesenteric artery: A complication in the management of a type II endoleak

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The optimal management of endoleaks after endovascular repair of abdominal aortic aneurysms remains to be established. In this report, we describe a persistent side-branch, or type II, endoleak 1 year after endograft implantation treated with catheter-directed embolization of the aneurysm sac and the inferior mesenteric artery via the superior mesenteric artery, with embolization agents including thrombin, lipiodol, and Gelfoam powder. Shortly after the embolization procedure, colonic necrosis developed in the patient, manifested by peritonitis, which necessitated a partial colectomy. This case underscores the devastating complication of colonic ischemia as a result of catheter-directed embolization of the inferior mesenteric artery in the management of an endoleak. (J Vasc Surg 2001;34:1119-22.)

The goal of endovascular abdominal aortic aneurysm (AAA) repair is to prevent aneurysm rupture by means of excluding the aneurysm from the aortic circulation using the stent-graft device. Several reports noted that 8% to 44% of patients have a persistent flow within the aortic aneurysm sac, also known as an endoleak, despite a technically successful endovascular AAA repair.[1-3] The presence of an endoleak implies potential pressurization of the AAA, which, if left untreated, may lead to aneurysm enlargement and subsequent rupture. A type II endoleak is characterized by the persistent flow of side-branch vessels that perfuse the aneurysm sac, with the inferior mesenteric artery (IMA) and lumbar arteries being the most common side-branch vessels.[1,2,4] We herein report a patient with a type II endoleak 1 year after endovascular AAA repair who underwent a transcatheter embolization of the aneurysm sac and the IMA in an effort to eliminate the endoleak, which resulted in colonic necrosis. The possible causes of such a complication as well as lessons we learned from this experience are discussed in this report.

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CASE REPORT

A 78-year-old man with a diagnosis of a 3.6-cm AAA 3 years earlier was noted to have an enlarging aneurysm of 4.8 cm on a surveillance ultrasound scan that was confirmed by computed tomography (CT). Because of a family history of aortic aneurysm rupture and evidence of aneurysm enlargement, the patient was evaluated for an endovascular AAA repair and was deemed a suitable candidate for aortic endograft placement. He underwent an uneventful endovascular grafting using a modular bifurcated stent-graft device (Excluder, W. L. Gore and Associates, Inc, Flagstaff, Ariz) in June 1999. No endoleak was identified on completion angiography or spiral CT at the time of discharge. However, the 1-month, 6-month, and 12-month follow-up CT scans demonstrated contrast extravasation into the AAA sac with no increase in aneurysm diameter (Fig 1). Because of the persistent endoleaks, the patient underwent a diagnostic and therapeutic aortogram in an effort to identify and eliminate the endoleak 13 months after the endovascular stent-grafting procedure. Via a right femoral artery access, the patient's superior mesenteric artery (SMA) was cannulated using a 0.035-in Bentson guidewire (Boston Scientific Vascular/Medi-tech, Oakland, NJ) and a Barenstein catheter (Medi-tech). Next a coaxial microcatheter (Fast Tracker, Boston Scientific Vascular, Natick, Mass) was used to gain access to the aneurysm sac from the SMA through the IMA by means of the marginal artery. A combination of thrombin solution (2000 units; Gentrac, Middletown, Wis), lipiodol (2 mL; Therapex, Montreal, Quebec), Gelfoam powder (1 g, Pharmacia & Upjohn Co, Kalamazoo, Mich) was introduced into the sac via the microcatheter (Fig 2). The catheter system was then withdrawn into the origin of the IMA for embolization. Although completion angiography demonstrated no flow into either the aneurysm sac or the IMA, the sigmoidal arteries were noted to be thrombosed (Fig 3).

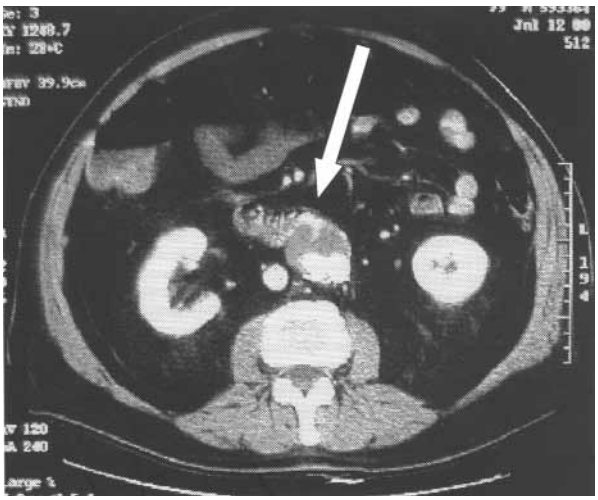

Fig 1. Abdominal CT scan demonstrating an endoleak (*arrow*) after the stent-graft placement.

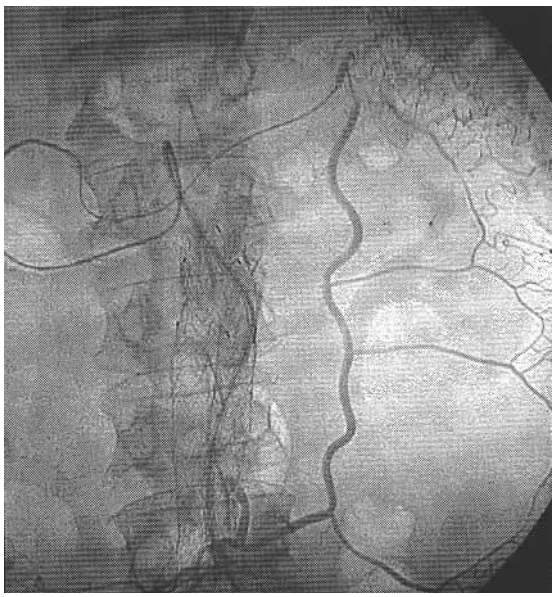

Fig 2. A microcatheter was used to cannulate the SMA, traverse the IMA via the middle colic artery, and finally reach the aneurysm sac for thrombin and Gelfoam embolization.

Six hours after the procedure, back pain and mild lower abdominal pain developed in the patient. The abdominal pain progressed to severe guarding with mild rebound tenderness. The patient developed a fever of 102.2°F and a leukocytosis of 30,000. Diagnostic laparoscopy revealed a necrotic sigmoid colon and was followed by an exploratory laparotomy and a left hemicolectomy and transverse colostomy placement. The patient recovered without further incident and was discharged to a rehabilitation center on postoperative day 15. Abdominal CT scanning 3 months later showed no evidence of an endoleak, and the aneurysm diameter remained unchanged.

DISCUSSION

Incomplete exclusion of an AAA after implantation of an endovascular graft has been reported to occur in 8% to 44% of all cases, which poses a potential threat of continuous aneurysm expansion and thus constitutes a failure of endoluminal repair.[1-3] The optimal timing of intervention and the methodology of the treatment of an endoleak remain challenging and often controversial issues. Concern is for continued pressurization of the aneurysmal sac with subsequent rupture. Retrograde flow via branch vessels such as the IMA or lumbar arteries is responsible for the majority of type II endoleaks, requiring the elimination of flow to protect the aneurysm from possible enlargement.[4-6] There is a general consensus that persistent endoleaks leading to aneurysm expansion necessitate treatment by either operative or endovascular intervention.[6-8] However, in the presence of a type II endoleak without aneurysm expansion, the timing and indication for intervention remain controversial.[6-8]

Despite the lack of a demonstrable aortic aneurysm enlargement in our patient, his persistent endoleak 1 year after the endograft placement prompted us to intervene. Because the CT of the abdomen showed a persistent flow in the IMA and the intraoperative angiogram confirmed the absence of either proximal or distal attachment endoleak, we were certain that a type II endoleak was present in our patient. It is noteworthy that a type II endoleak implies the presence of at least two side branch vessels that provide a continuous flow circuitry within the aneurysm sac. Because the aortogram only identified a patent IMA in our patient, we postulated that a second side branch vessel was present but not visible by currently available diagnostic imaging modalities. We therefore chose to perform a catheter-directed embolization of both the IMA orifice and the aneurysm sac via the SMA using liquid embolic materials that included lipiodol, thrombin, and Gelfoam. We hypothesized that by administering these liquid agents in the aneurysm sac and IMA orifice, the diffuse permeation of these materials could effectively induce thrombosis within the aneurysm sac along with any remaining patent side branches. In contrast to catheter-directed coil placement, the liquid property of these embolic materials provided a theoretical advantage that more diffuse aneurysm sac thrombosis could be achieved without packing a large amount of coils in the aneurysm sac.

The Gelfoam and thrombin were used as embolic agents because of their prothrombotic properties. Lipiodol was also chosen because it is a highly visible oil that facilitated the visualization of the embolization process. The inclusion of lipiodol raises a potential concern because of its radioopacity, which may interfere with future endoleak detection. This issue is overcome by the availability of new CT computer software (ImageWare, Inc, Atlanta, Ga) that permits subtraction of a pre-existing contrast-enhanced density on a CT scan (such as lipiodol), thus allowing future detection of a new contrast-enhanced

density (such as a new endoleak). The mixture of Gelfoam, thrombin, and lipiodol resulted in a liquid embolization fluid that became rather unpredictable in its ability to remain localized within the aneurysm sac or the IMA orifice after catheter-directed infusion. These agents were extruded by hand injection through a syringe into the aneurysm sac via the subselective catheterization of the IMA via the SMA. After that, the catheter system was slowly withdrawn, and IMA origin was embolized using the liquid embolic materials. The manipulation of the catheter containing embolization agents at this point raised several concerns. First, particles or fragments that initiate thrombosis by forming a gelatinous mass may lodge in and occlude the catheterized artery and its larger branches. During catheter removal, minute fragments may potentially remain in the collateral arcades between the SMA and IMA, leaving these important collaterals inadvertently thrombosed by the trailing embolization material. Indeed, the completion angiogram demonstrated occluded sigmoidal artery branches, presumably a result of inadvertent migration and embolization of the embolic agents, which undoubtedly contributed to our patient's colonic necrosis.

The use of catheter-directed infusion of gelatin sponge fragments, thrombin, or polyvinyl alcohol has been described previously in the management of gastrointestinal hemorrhage.[9,10] A major disadvantage of particulate materials is bowel infarction from distal migration, especially with very small particles.[11] Even though transcatheter embolization has been reported to be a definitive and effective means of treating patients with lower gastrointestinal hemorrhage, intestinal ischemia and infarction have occurred in as many as 20% of cases, limiting this procedure to being recommended for high-risk patients who are not deemed candidates for emergency surgery.[9,10] In retrospect, the application of thrombin and Gelfoam in the IMA in our patient was a hazardous maneuver, given the high incidence of bowel infarction derived from the gastrointestinal hemorrhage literature.

Recent literature has demonstrated the safety and efficacy of coil embolization of the perigraft space and outflow vessels.[3,5,12,13] With this treatment option, tight coil packing of endoleaks is thought to be important in achieving solid thrombus formation and preventing further flow into the aneurysm sac. Furthermore, oversizing the coil should lessen the likelihood of migration once it is deployed within a vessel or the aneurysm sac, which could have avoided the inadvertent sigmoidal artery occlusion seen in our patient. Whereas it is probable that catheter-directed coil placement in the aneurysm sac and the IMA in our patient could have avoided the devastating complication of colonic necrosis, this option was not chosen in our patients because of several reasons. First, it would require an enormous number of coils to fully pack the aneurysm sac to achieve a complete aneurysm sac thrombosis. Second, catheter manipulation via the mesenteric vessel for coil placement within the aneurysm sac would be technically challenging to ensure that coils are adequately placed throughout the aneurysm sac. And last, placement of a large number of coils may render subsequent endoleak evaluation difficult because of coil-related artifacts on future CT of the abdomen.

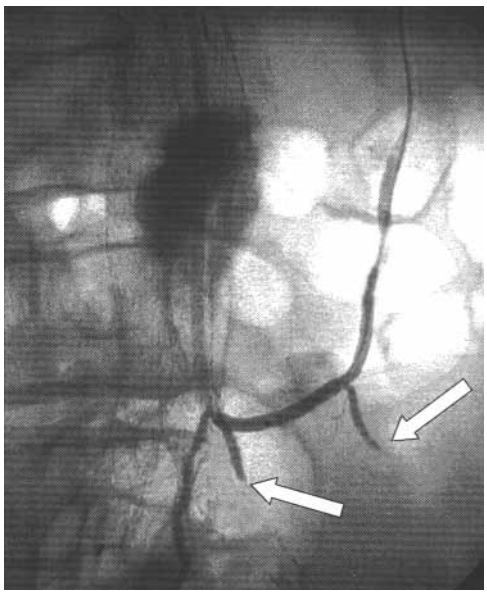

Fig 3. After embolization of the aneurysm sac and the IMA orifice, the completion angiogram demonstrates radio-opaque lipiodol mixture in the aneurysm sac with occluded sigmoidal artery branches (*arrows*).

An alternative treatment for type II endoleaks is that of retroperitoneal endoscopic ligation of the IMA and the lumbar arteries.[14] In addition, percutaneous translumbar embolization of the aneurysm sac or placement of thrombogenic sponges in the aneurysm sac have been reported to be feasible in anecdotal reports.[15,16] Unknown, however, is the appropriate timing of a secondary endovascular intervention, because a significant percentage of type II leaks will seal spontaneously after stent-graft implantation. The length of time that an endoleak may be safely observed has not been determined and may depend on the degree of aneurysm expansion. Given the relative stable aneurysm diameter of 4.8 cm in our patient despite the persistent type II endoleak, continuous surveillance may remain a viable alternative. Under such a circumstance, intervention of the endoleak should be considered when the aneurysm displays evidence of enlargement.

In summary, we described a case of persistent type II endoleak 1 year after endovascular AAA repair. With selective IMA catheterization via the SMA, various embolization agents including thrombin and Gelfoam were infused into the aneurysm sac and IMA orifice. With the resultant colonic necrosis caused by inadvertent sigmoidal artery embolization, the intervention performed in our patient was clearly hazardous and undoubtedly contributed to his catastrophic complication. We strongly recommend that such a maneuver be avoided in the management of a type II endoleak.

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