

Use of glue and microcoils for transarterial catheter embolization of a type 1 endoleak

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ABSTRACT

We present a unique case of abdominal aortic aneurysm initially presenting with inferior vena cava compression leading to deep venous thrombosis, for which the patient subsequently underwent an endovascular aortic repair. Aorto-uni-iliac endografting was performed for subacute occlusion of left common iliac artery complicated by proximal type 1 endoleak. Subsequent management of the endoleak was successful, using a liquid embolic agent (cyanoacrylate) by transarterial approach. Transarterial catheter embolization with glue and coils is a feasible technique for high flow type 1 endoleaks. Glue injection carries the risk of non-target embolization, and thus this option should be reserved for experienced hands.

Key words: • endovascular aneurysm repair
• abdominal aortic aneurysm • endoleak • embolization
• deep venous thrombosis

Endovascular management of abdominal aortic aneurysms (AAA) is an evolving alternative to open surgical repair. Although this less-invasive method has promising results, endoleaks remain an important complication (1). High-flow proximal type 1 endoleaks, resistant to balloon dilation, present a risk of enlargement and rupture of the aneurysmal sac (2). In this study, we report a case of a giant AAA presenting with compression of the inferior vena cava, leading to deep venous thrombosis (DVT) and extensive pulmonary thromboemboli (PTE). The patient was treated with an aorto-uni-iliac endografting procedure for a subacute occlusion of the left common iliac artery. Warfarin treatment for DVT and PTE, in conjunction with a persistent type 1 endoleak, resulted in recanalization of the left common iliac artery. Treatment of the type 1 endoleak was performed on day 10, with successful coil and glue embolization of the aneurysmal sac via a left femoral artery approach.

Case report

A 70-year-old man with history of coronary artery disease, hypertension, and hyperlipidemia was admitted to the emergency service with a complaint of swelling of his right leg. On physical examination, edema of the right leg, hypothermia of the left leg, and lack of left popliteal pulse were noted. Physical examination of the abdomen revealed a pulsatile mass which was visible on the anterior abdominal wall. On admission, blood laboratory studies were unremarkable. ECG and cardiac enzymes were within normal limits. Pulse oximetry showed mild desaturation (92%) in room air. Doppler ultrasound examination of lower extremities revealed bilateral thrombosis of common femoral veins. Contrast-enhanced thoracic computed tomography (CT) was performed in order to evaluate for PTE. Reformatted CT angiography (CTA) images demonstrated diffuse thromboemboli within the main, lobar, and segmental branches of both pulmonary arteries. An abdominal series obtained for DVT revealed an incidental finding of a giant fusiform infrarenal AAA (12.8 x 11 cm maximum dimensions) originating 3 cm distal to the left renal artery and extending to the terminal aorta (Fig. 1). The right common iliac artery showed fusiform dilatation, and the left common iliac artery was occluded. Extensive collateral vessels supplied the left external iliac artery. Although anticoagulation was indicated for DVT and PE, this treatment was contraindicated because of the risk of rupture of the AAA. A decision was made to place a vena cava filter, with a diagnostic arteriogram to evaluate the AAA and iliac arteries. Using a right internal jugular vein approach, inferior vena cavography was performed, revealing severe compression of the infrarenal inferior vena cava by the giant AAA, which was thought to be the etiology of the bilateral DVT. Following the diagnostic procedure, an OptEase permanent/retrievable vena cava filter (Cordis, War-

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Figure 1. Reformatted CT angiography image. Large abdominal aortic aneurysm with significant neck angulation and mural thrombus (*asterisk*). Additionally, left common iliac artery is occluded (*arrow*).



Figure 2. Day 10 CT angiography. 3D reconstruction image showing type 1 endoleak (*small arrow*) and partially recanalized left common iliac artery (*big arrow*) as well as the inferior vena cava filter (*arrowhead*).

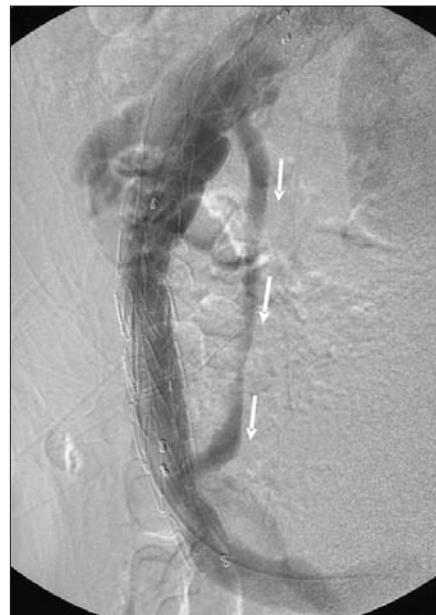


Figure 3. Day 11 abdominal aortogram showing proximal type 1 endoleak recanalizing the left common iliac artery, steep oblique image. Arrows indicate the route of the endoleak.

ren, New Jersey, USA) was placed in the suprarenal inferior vena cava at the level of hepatic veins via the same venous access. Subsequent conventional aortogram findings were consistent with CT findings with regard to the left common iliac artery stump. Furthermore, the right external iliac artery was found to be significantly angulated, with a maximum diameter of 5.5 mm. Endovascular aneurysm repair was scheduled for the next day. Because the left common iliac artery occlusion was asymptomatic, aorto-uni-iliac endograft was the treatment of choice. On the following day, the infrarenal AAA was excluded from the circulation by placing a 34-16 x 170 mm Medtronic Talent (Medtronic INC, Minneapolis, Minnesota, USA) aorto-uni-iliac endograft. The left re-

nal artery was protected and the graft was extended distally to the origin of the right common iliac artery. Later, a second 18-14 x 140 mm endograft Medtronic Talent iliac extension (Medtronic INC, Minneapolis, Minnesota, USA) was deployed within the first one. The right internal iliac artery was protected during deployment of the second graft. Late phase images of control angiograms revealed a minimal proximal type 1 endoleak. By using a Reliant balloon catheter (Medtronic INC, Minneapolis, Minnesota, USA), balloon angioplasty was performed to the most proximal infrarenal landing zone in order to obtain a better seal with the endograft. However, there was still some minor proximal type 1 endoleak after the ballooning. The patient was then anticoagu-

lated (Coumadin® 10 mg, Aspirin® 100 mg) for DVT and PTE, despite the minimal type 1 endoleak. He was discharged home in good condition 4 days after the procedure. Because the lumbar collaterals supplying the left external iliac artery were occluded by the endograft, patient consent was obtained for possible future femoro-femoral bypass.

On postprocedural day 10, control CTA demonstrated patency of the aorto-right-uni-iliac tubular endograft as well as increased proximal type 1 endoleak. Additionally, the left common iliac artery was seen to be partially re-canalized, most likely secondary to warfarin treatment and the high-flow endoleak (Fig. 2). However, there was no increase in aneurysmal sac diameter. Because of the ongoing risk of rupture of the aneurysm due to the endoleak, and the need for anticoagulation, the patient was readmitted to the hospital one day after the control CTA for further management of the endoleak. Under IV sedation, and using a right common femoral artery approach, a pig-tail catheter was placed at the level of the renal arteries. Biplane abdominal aortogram showed an extensive proximal type 1 endoleak recanalizing the left common iliac artery (Fig. 3). Under



Figure 4. Lateral aortogram showing the glue cast as well as microcoils along the course of the endoleak.



Figure 5. Final aortogram showing patent aorto-uni-iliac graft with no endoleak.



Figure 6. Sixth-month control axial CTA image at arterial phase showing patent endografts with no endoleak. Coil pack causing striking artifact, and the glue cast at the posterior aneurysmal sac (*arrow*) can be seen.

sonographic guidance, a 6F introducer sheath was placed through the left common femoral artery into the left external iliac artery. After visualizing the exact course of the proximal type 1 endoleak through the partially recanalized, a 4F catheter was advanced into the caudal portion of the sac, followed by placement of a coaxial microcatheter (Rebar 18, Micro Therapeutics, INC, Irvine, California, USA) within the proximal endoleak cavity just below the level of renal arteries. Through the microcatheter, multiple detachable platinum coils (Micrus

platinum microcoil system, Micrus Corporation, Sunnyvale, California, USA) were placed into the sac in order to construct a frame and to diminish blood flow, thus providing a safer milieu for the injection of glue. Next, an 18% n-butyl 2-cyanoacrylate (NBCA) (Histoacryl, B. Braun, Tuttlingen, Germany) and Lipiodol (Lipiodol ultrafluide, Laboratoire Guerbet, Aulnay-Sous-Bois, France) mixture was injected, initially into the anterior portion of the sac, and subsequently into the posterior portion as the catheter was withdrawn during the injection. The

sac was thus embolized sequentially from anterior to posterior. The resultant glue cast compromised the origin of the endoleak just below the level of renal arteries (most cephalic portion), and extended to the origin of the left common iliac artery postero-inferiorly (Fig. 4). Control aortogram revealed total obliteration of the endoleak (Fig. 5). Additionally, it was noted that left external iliac artery was supplied by the contralateral internal iliac artery. Control CTA on postprocedural day 3 demonstrated total occlusion of the endoleak. One day later, the patient was discharged without complications. Six months after the procedure, control CTA demonstrated stable sac size; no endoleak was seen (Fig. 6). At one year clinical follow up visit, the patient was doing well.

Discussion

One of the most feared complications of the endovascular aneurysm repair is graft-related endoleaks that are associated with increased risk of rupture (3, 4). Endoleak is defined as the persistent blood flow within the aneurysmal sac after endovascular graft deployment (5). White et al. classified endoleaks according to the site of the blood flow into the aneurysmal sac (6). Type 1 endoleak is defined as the inadequate seal at the proximal or distal extremities of the endograft (7). One multicenter study of endograft deployment for infrarenal AAA reported endoleak in 12.3% of patients (8). In other studies, endoleak has been observed in 15–21% of clinical trials involving commercially produced endovascular stent grafts (9). Type 1 leaks (those occurring at the proximal end of the graft) are the most likely to rupture (3). Endovascular management, such as proximal stent-graft extension or open surgical repair is the main option for treatment of type 1 endoleaks (9). Another option is conservative management by stopping anticoagulation and observing the endoleak until it thromboses spontaneously. In this situation, the risk of rupture may increase gradually (10). Selection of treatment method for the endoleak depends on the anatomy of the endoleak and the health status of the patient. Becquemin et al. treated type 1 endoleaks in their series by adding new pieces of endograft at the site of leaks, but they proposed that

this method was highly risky since putting a new graft in the infrarenal neck might block the renal arteries if the new graft was too high, and might fail to treat the endoleak if placed too low (11). In a study of Thomas et al., 15 out of 54 type 1 endoleak cases were treated by proximal cuff or a second stent-graft (12). Of those 15 cases, only one patient died from aneurysmal rupture. Three patients had percutaneous transluminal angioplasty, which was successful in two of them. In 17 patients, immediate surgical repair was affected. Of those, five patients died, and two had persistent endoleak. The remaining patients had no treatment for endoleak. Coil embolization is used mainly for type 2 endoleaks (those originating from collateral branches of the aneurysmal sac) (9). Liquid embolizing agents such as n-butyl-2-cyanoacrylate (NBCA) and Onyx[®] are also used for the treatment of type 2 endoleaks (5, 13). Traditionally, coil and/or glue embolization is not used for treatment of type 1 endoleaks.

Our case presented with DVT and massive PTE, which were secondary to compression of inferior vena cava and iliac veins by AAA. Few cases of AAA presenting with DVT are reported in the literature (14). The proposed mechanism is mechanical obstruction leading to venous stasis and ultimately to thromboemboli, as we observed in our patient. In elderly patients presenting with unexplained DVT, the presence of AAA and/or an iliac artery aneurysm should be kept in mind as the underlying etiology. Since anticoagulation was contraindicated in our case, our plan was to insert a prophylactic inferior vena cava filter at the suprarenal level in order to prevent progression of PTE.

In our patient, the endovascular aneurysm repair was performed by an aorto-right uni-iliac tubular endograft because the left common iliac artery was occluded, and only one iliac artery was suitable for access and graft deployment. Aorto-uni-iliac endografting is a preferred method in treatment of AAA under certain conditions, such as ectasia or significant aneurysmal dilatation of the iliac artery, as well as tortuosity or occlusion that may lead to inadequate graft-vessel landing zones or difficulty in placing the graft. Under such circumstances,

iliac limitation of graft deployment can be overcome by aorto-uni-iliac graft with femoro-femoral crossover by-pass grafting (15). In our patient, lumbar collaterals and contralateral hypogastric arteries initially supplied the left external iliac artery. Despite the occlusion of lumbar collaterals by the stent-graft, our patient did well after the endovascular aneurysm repair and subsequent endoleak treatment, except for mild to moderate left leg claudication. Our plan was to perform a femoro-femoral bypass if ischemia of the left leg were to develop. Control CTA on day 10 showed type 1 endoleak and spontaneous re-canalization of left common iliac artery most likely secondary to anticoagulant therapy for DVT and PTE in addition to the type 1 endoleak. Graft extension by proximal second graft implantation could not be performed because the proximal endoleak was too close to the origins of renal arteries. Moreover, co-morbidities and the age of the patient weighed heavily against open surgical repair. Also, the patient's situation required intervention because the aneurysmal sac was compressing the inferior vena cava and iliac veins and was causing DVT and PTE. This particular patient was at high risk for a type 1 endoleak (as was predicted prior to the procedure) due to the extremely angulated infrarenal neck (Fig. 1). We performed a balloon angioplasty to the infrarenal portion of the endograft as the first step in management of the proximal type 1 endoleak. Then, transarterial embolization was performed in order to treat the type 1 endoleak. Since the flow was so fast within the sac, detachable microcoils were used initially to construct a frame; then diluted glue was injected to fill the sac. Diluted glue was preferred embolizing agent due to its adjustable polymerization time and optimum radiopacity during the procedure. Of course, even in experienced hands, the use of a liquid embolizing agent carries the risks of reflux and non-target embolization. In order to avoid those complications, we were extremely careful with our injection. Accordingly, we employed coils and a sheath-catheter system within the left common iliac artery in order to avoid distal non-target embolization. In addition, aortic blood flow protected the renal arteries from the risk of reflux. The literature contains

very few reports of treatment of type 1 endoleak by a liquid embolic agent via transcatheter approach (16, 17).

In conclusion, abdominal aortic aneurysm may present with DVT and/or PTE. In selected cases, transarterial embolization for the treatment of type 1 endoleaks can be performed safely (especially in experienced hands) by using coils and glue.

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