Surgical access of the gluteal artery to embolize a previously excluded, expanding internal iliac artery aneurysm

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We describe open exposure of the inferior gluteal artery to allow coil embolization on an enlarging internal iliac artery aneurysm after previous abdominal aortic aneurysm (AAA) repair. An 84-year-old man with a stoma had undergone open AAA repair surgery 8 years previously, during which the proximal aortic neck and both proximal external iliac arteries were ligated, followed by an aorta to right external iliac and left common femoral bypass. Eight years later, he complained of abdominal pain, and a computed tomographic (CT) scan revealed persistent flow in the right internal iliac artery with enlargement to 8 cm in diameter. Because prograde access to the internal iliac artery was not possible as a result of the previous exclusion, the inferior gluteal artery was exposed surgically. Coil embolization of the arteries supplying the internal iliac artery aneurysm was successfully performed. The AAA and internal iliac artery aneurysm were treated by the exclusion technique. Eight years after the operation, CT revealed that the iliac artery had expanded to approximately 8 cm in diameter. The patient was placed face down, and a catheter was directly inserted into the internal iliac artery from the inferior gluteal artery. Four embolization coils were placed in the internal iliac artery and its branches. Absence of blood flow and shrinkage of the aneurysm were subsequently confirmed in the aneurysm, as shown by echogram color duplex scanning and CT scanning at 1 year. This technique could also be applicable for persistent blood flow in an internal iliac aneurysm after endovascular AAA repair, and the size of the aneurysm was reduced to approximately 1 cm 1 year after the operation. (J Vasc Surg 2007;45:387–90.)
was identified below the piriformis muscle (Fig 2, a). A 5F introducer sheath was inserted into the inferior gluteal artery by using the Seldinger technique, and then the distal inferior gluteal artery was ligated. An angiogram showed a right common and internal iliac artery aneurysm and its branches (Fig 2, b). After angiography, by using a directional catheter, embolization coils (20 × 200 mm and three coils of 15 × 150 mm; Tornado Embolization Microcoils; Cook, Bloomington, Ind) were inserted into the aneurysm and the feeding arteries, including the obturator artery and the inferior gluteal artery. Completion angiogram confirmed the ab-
sence of blood flow in the aneurysm (Fig 2, c). Contrast CT scan obtained 2 weeks after the embolization procedure revealed complete thrombosis of the aneurysm. This was also confirmed with a transabdominal duplex scan. The postoperative course was uneventful. The patient did not experience buttock claudication or any ischemic complications. A follow-up CT scan obtained 1 year after the procedure showed shrinkage of the aneurysm by 1 cm and also an absence of blood flow (Fig 3).

DISCUSSION

Surgical exposure and control of the distal internal iliac artery are sometimes difficult. In our case, an 8-cm aneurysm had developed in the internal iliac aneurysm that was excluded from the circulation with proximal ligation 8 years before presentation. Surgical access was believed to be difficult because of the presence of a colostomy and the two previous laparotomies. Coil embolization is an alternative to surgical repair; however, in this case, catheter-based access in a standard fashion (prograde) to the aneurysm was not possible because the aneurysm had already been excluded previously. The inferior gluteal artery can be detected with a duplex scan and can serve as an access vessel for intervention of the internal iliac artery and its branches. In this case, the operation was conducted with the patient under general anesthesia, and access was obtained via a surgical cutdown. However, percutaneous duplex-guided access seems feasible.

During coil embolization of an internal iliac artery, whether it be done in a prograde or retrograde manner, distal embolization may occur to the branches of the internal iliac artery as well as the lower extremities. Such embolization can cause necrosis of the buttock muscles and skin, and, therefore, coil embolization should be performed with care, with attempts to occlude the arteries as proximally as possible.

As far as the risk of ongoing perfusion of a previously excluded aneurysm is concerned, Darling et al reported that 4% of excluded AAAs had persistent blood flow in the aneurysm sac and that, of these, 0.6% went on to rupture. Thus, we believed that it was reasonable to treat this large iliac aneurysm. The presence of blood flow in the aneurysm was seen with a higher frequency in patients who were receiving anticoagulation therapy. However, our patient was not taking chronic anticoagulation therapy. The reason why flow persisted in our patient may be related to the fact that the inferior and superior gluteal arteries and the obturator artery served as inflow and outflow.

Our case is analogous to a type II endoleak after endovascular aneurysm repair. Although the clinical significance and the risk of a type II endoleak are controversial, several reports have suggested that rupture can occur from type II endoleaks. For example, Bade et al reported on a rupture of an excluded hypogastric aneurysm after endovascular aneurysm repair.

Although we were able to obtain complete thrombosis of the aneurysm with coils, long-term follow-up imaging studies are needed. This is because, as pointed out by Marty et al, the absence of endoleak on angiographic and CT scans does not always mean that the aneurysm has been depressurized effectively.

In summary, we report a novel access to the internal iliac artery via the inferior gluteal artery. This approach may be especially valuable in cases in which prograde access to the internal iliac artery is difficult or impossible, including persistent flow in an internal iliac artery after exclusion during endovascular aneurysm repair.

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REFERENCES


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