Successful percutaneous transgluteal embolization of a complex arteriovenous malformation feeding a hypogastric artery aneurysm

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ABSTRACT

Pelvic arteriovenous malformation (AVM) is a rare condition mostly requiring a complex therapeutic strategy. The surgical approach is challenging and burdened by relatively high mortality and morbidity rates. No guidelines are available for the endovascular treatment of AVM because the literature is limited to small case series and case reports. We present a complex case of a pelvic AVM associated with an internal iliac artery aneurysm in a patient previously treated with a common to external prosthetic substitution for aneurysm and proximal ligation of internal iliac artery. (J Vasc Surg Cases and Innovative Techniques 2018;4:45-9.)

An arteriovenous malformation (AVM) consists of multiple anarchic communications between the arterial and venous systems. Pelvic AVM is a rare but extremely distressing condition and represents 1.8% of the major abdominal AVM localizations. Its treatment, either open or endovascular, is challenging because of the anatomic characteristics, the deep location, and the high hemorrhagic and ischemic risks.[1]

Pelvic AVM has different causes (idiopathic, post-traumatic, neoplastic, and congenital) and can be life-threatening during delivery or rupture (spontaneous or post-traumatic).[2,3] Iliac AVM is sometimes associated with a hypogastric artery aneurysm (HAA) and can lead to an arteriovenous fistula.[4] Because of the high hemorrhagic risk, surgical removal of an ilioiliac AVM is rarely indicated.[5] We present a complex case of a pelvic AVM associated with a right internal iliac artery aneurysm. The patient agreed to publication of the case.

CASE REPORT

A 70-year-old man was admitted to our hospital with pelvic pain and dysuria. The patient's history was silent with the exception of the occasional discovery of a pelvic congenital AVM. In 2008, he underwent common to external right iliac artery substitution for aneurysm and proximal ligation of the right hypogastric artery. The surgical strategy had probably been chosen for the patient's relatively young age, the moderate dilation (15 mm) of the first tract of the right hypogastric artery, the absence of a proper hypogastric distal landing zone, and the presence of the AVM (higher risk of perioperative bleeding during surgical distal hypogastric isolation). On postoperative duplex ultrasound follow-up, we observed a slow but progressive growth of a right HAA and a concomitant growth of the pelvic AVM, probably related to the hyperperfusion of the collateral branches of the hypogastric artery. In 2016, the patient underwent computed tomography angiography that confirmed the presence of a 48-mm right HAA fed by a voluminous pelvic AVM (Fig 1). Digital subtraction angiography was performed, demonstrating multiple AVM afferent vessels feeding the HAA from lumbar arteries, contralateral hypogastric branches, and the ipsilateral inferior epigastric artery (Fig 2).

The presence of a hostile abdomen, the deep location of the AVM, and the high surgical risk led to the choice of an endovascular approach that was, however, extremely challenging because of the previous ligature of the origin of the hypogastric artery.

The procedure was performed under local anesthesia (10 mL of lidocaine hydrochloride 1%) in a hybrid operating room with appropriate precautions in case of rapid open conversion. The patient was placed in prone decubitus position, and an ultrasound-guided direct puncture of the right gluteal artery was performed (Fig 3). The right sciatic nerve was visualized first, then the right gluteal artery was detected in a medial and slightly more superficial plane. A 4F 45-cm-long sheath (Flexor Check-Flo; Cook, Bloomington, Ind) was inserted on a 0.35-inch guidewire (angled Glidewire; Terumo, Tokyo, Japan) into the right HAA. Angiographic control confirmed the presence of the AVM with a rapid washout of the lesion through iliac veins (Fig 4). To avoid the risk of venous embolization and to reduce the AVM outflow, we decided to occlude the draining internal iliac vein with a 27-mm balloon occlusion catheter (Equalizer; Boston Scientific, Marlborough, Mass) delivered into the origin of the right internal iliac vein; we used a left jugular vein access,

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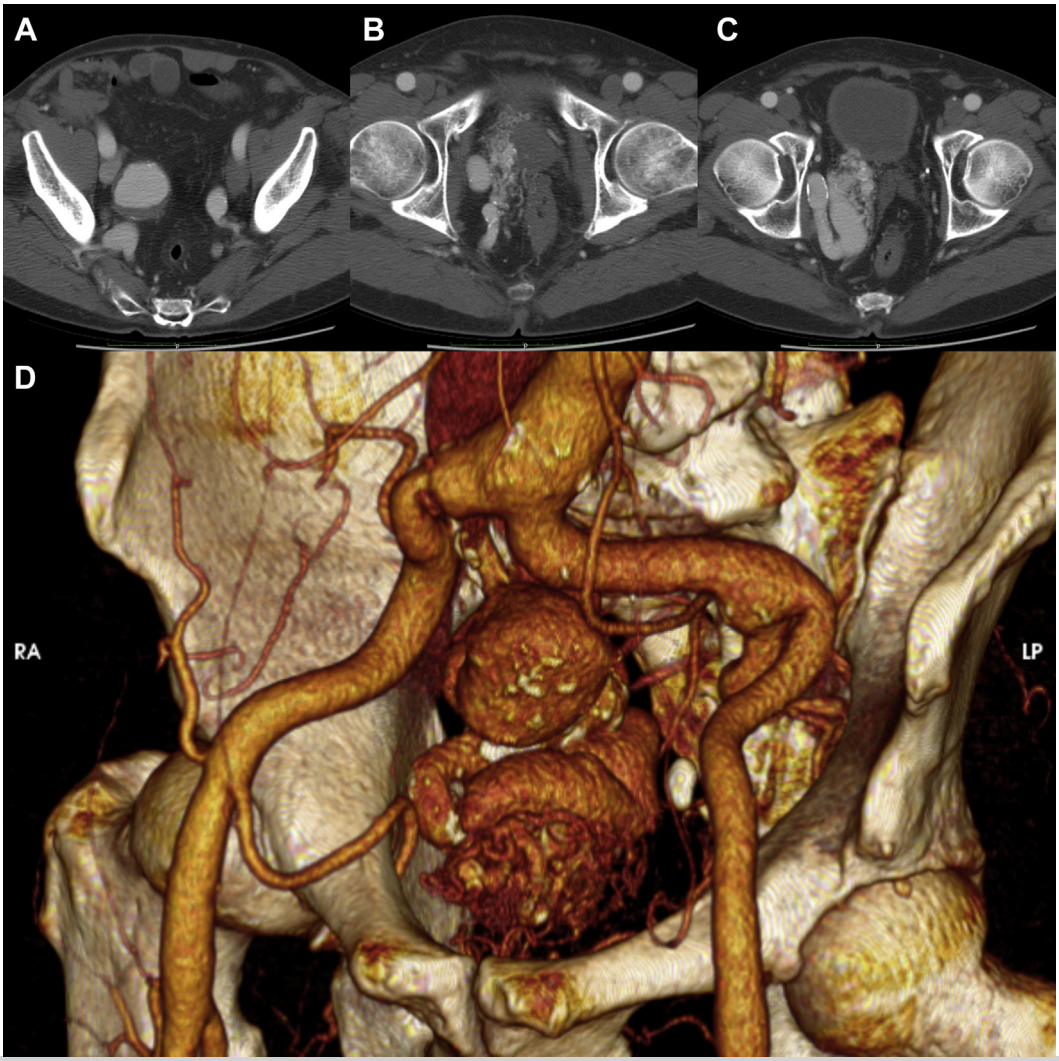

Fig 1. A-C, Computed tomography axial scans show a pelvic arteriovenous malformation (AVM) feeding a hypogastric artery aneurysm (HAA). **D,** Three-dimensional reconstruction of the pelvic AVM.

under ultrasound guidance, with the patient in prone position, the back slightly hyperextended, and a moderate hyperextension and left rotation of the head (Fig 3). After balloon inflation and digital subtraction angiography from the sheath in the right HAA, we proceeded to AVM branch embolization through a 4F angiographic catheter (Berenstein, Tempo; Cordis, Bridgewater, NJ) with a mélange 1:3 of 2 mL of acrylic synthetic surgical glue (Glubran 2; GEM, Viareggio Italy) and 6 mL of ethiodized oil (Lipiodol; Guerbet, Bloomington, Ind), followed by the placement of 0.35-inch metallic coils into the aneurysm sac (200 mm in length and 10 mm in diameter [MReye embolization coils; Cook] and 100 mm in length and 8 mm in diameter [Spirales Cirrus coils; BALT, Montmorency, France]). The angiographic control showed a partial occlusion of the AVM nidus with residual filling from contralateral hypogastric artery branches. We therefore decided to remove the gluteal access (after gluteal embolization and compressive medication), to turn the patient to the supine position, and to perform a new 5F access from the common left femoral artery (Fig 3). Multiple attempts of superselective catheterization (Cantata; Cook) of the left obturator artery feeding the AVM failed. From the same left femoral access, we engaged the right deep external pudendal artery, and we performed embolization of the remaining AVM feeding vessels with the same liquid embolic agent. Four hours after the beginning of the procedure, angiographic control showed complete exclusion of the AVM and the HAA (Fig 4). Because the procedure was free from complications, discharge occurred on the first postoperative day. At the 6-month follow-up, the patient was asymptomatic, and no adverse events had occurred. Computed tomography angiography confirmed the successful exclusion of the aneurysm with no AVM recurrence (Fig 4).

DISCUSSION

Open repair of AVM is a challenging procedure for vascular surgeons, with high mortality and morbidity rates of 25% to 30% and 12%, respectively.[1] Despite the

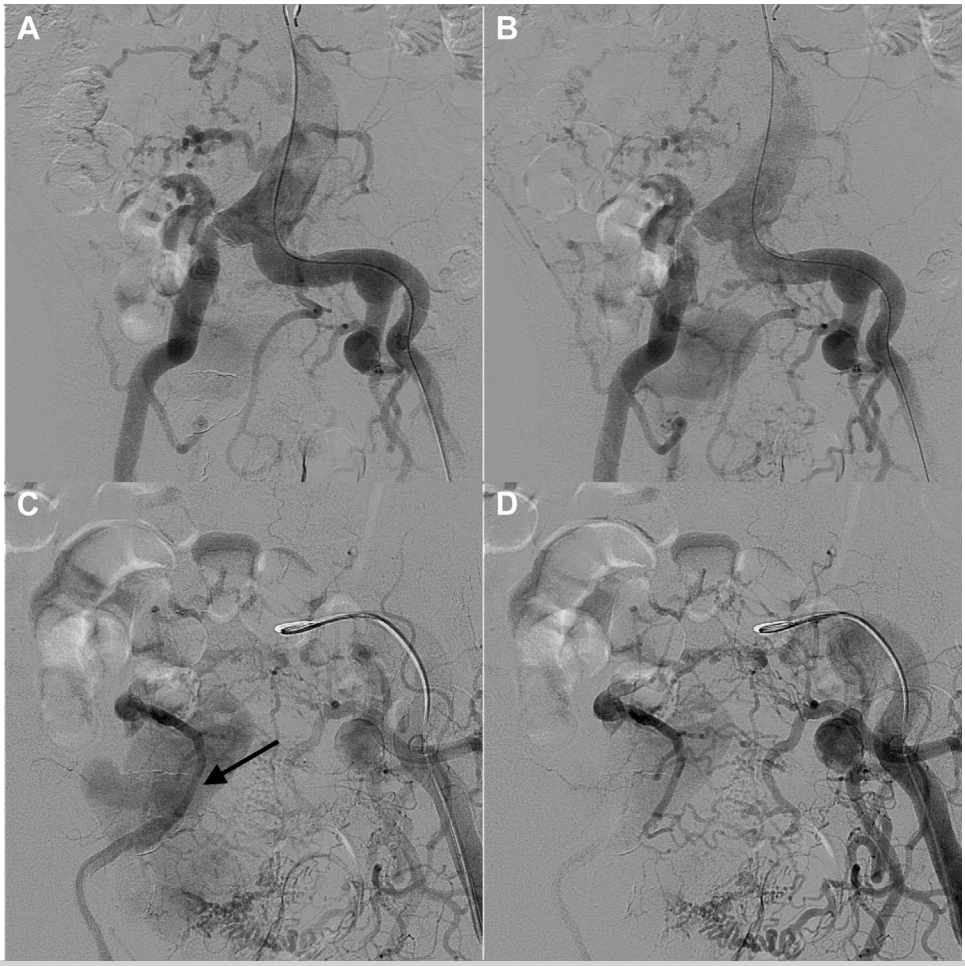

Fig 2. A and **B,** A posterior-anterior view of aortic angiogram shows hypertrophic right lumbar arteries and ipsilateral inferior epigastric artery feeding the arteriovenous malformation (AVM). **C** and **D,** Oblique views: hypertrophic contralateral hypogastric branches feeding the AVM. The *arrow* indicates the right external gluteal artery arising from the hypogastric aneurysm.

absence of guidelines on the endovascular technique for AVM embolization, the endovascular approach is the preferred treatment option because of its lower invasiveness, morbidity, and mortality and shorter hospital stay.[1-4,6,7]

In the literature, technical success is achieved in 94% of cases, and no perioperative mortality is reported.

Houbballah et al[6] proposed a hybrid treatment of pelvic AVM. A percutaneous embolization of the arterial feeding vessels was performed first, followed by an open access to the AVM with ligation of the efferent veins and direct embolization of the malformation. In a series of seven patients, the authors reported one pulmonary thromboembolism and two open reinterventions with no mortality.

Our decision was made to perform the whole procedure percutaneously to minimize the high surgical risk related to localization of the AVM, presence of the hypogastric aneurysm, and history of pelvic surgery. After accurate preoperative planning, the percutaneous transgluteal approach through the external gluteal artery was, according to us, the fastest and safest to reach the HAA and AVM nidus. Moreover, the venous balloon occlusion of the hypogastric vein guaranteed protection against distal embolization and a decreased washout effect, with consequent increased precision and efficacy of the procedure. The AVM embolization was performed with coils, acrylic synthetic surgical glue, and ethiodized oil because of the huge AVM size and the complex feeding vessel anatomy. This dilution and the injection of small boluses of glue allowed adequate delivery and rapid solidification with a reduction of the risk of distal embolization. Furthermore, the acrylic surgical glue currently has lower costs compared with other agents available on the market, even though new products are gaining favor in AVM embolization.[8-10]

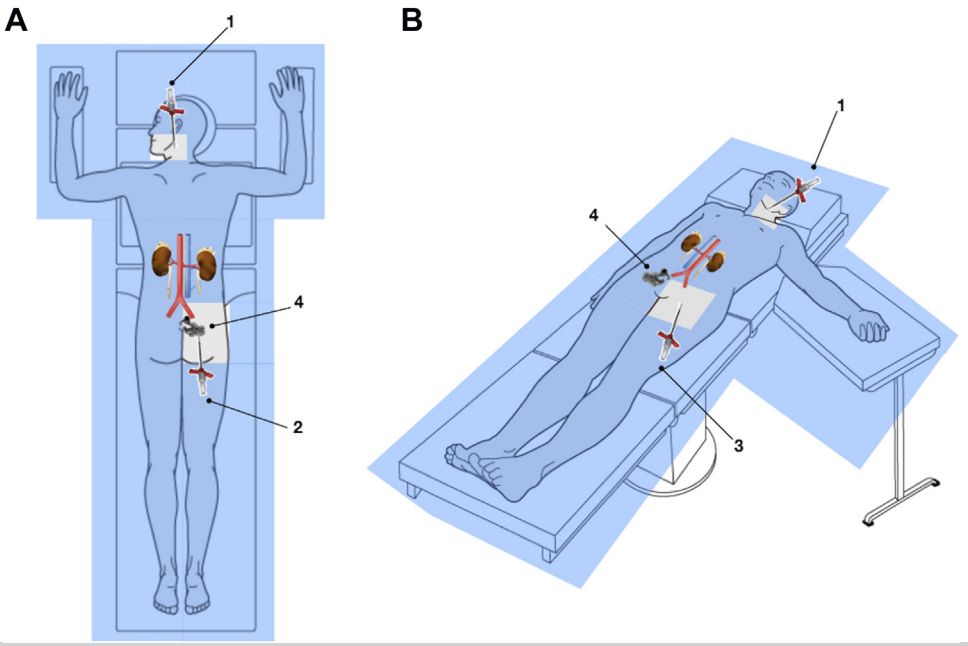

Fig 3. A and **B,** Diagrams showing placement of the patient and vascular percutaneous accesses at the beginning of the procedure and after repositioning. *1*, Left jugular access. *2*, Direct transgluteal access. *3*, Left common femoral artery access. *4*, Arteriovenous malformation (AVM) localization.

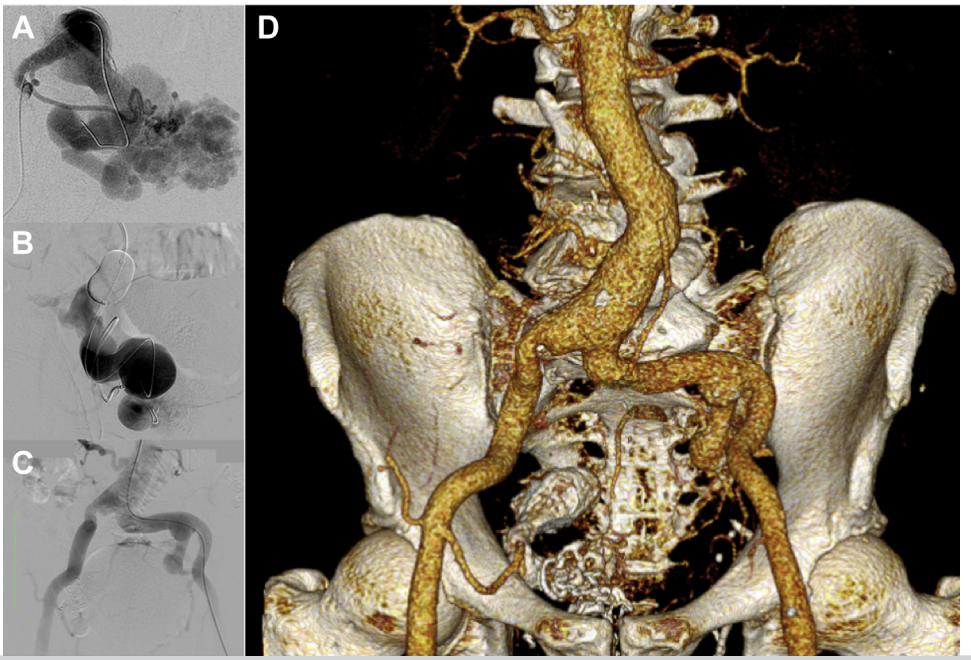

Fig 4. A-C, Intraoperative angiograms showing **(A)** direct puncture of the right gluteal artery, **(B)** inflation of an occlusion balloon in the right internal iliac vein, and **(C)** final control demonstrating complete exclusion of the arteriovenous malformation (AVM) and right hypogastric artery aneurysm (HAA) with coils and glue. **D,** Six-month three-dimensional reconstruction of computed tomography images showing the absence of hypogastric aneurysm reperfusion or recurrence of AVM.

CONCLUSIONS

In suitable anatomy, a complete full percutaneous endovascular approach to pelvic AVM can be safely performed. Venous balloon occlusion of the hypogastric vein can increase the efficacy of the procedure and avoid distal embolization, especially in high-flow AVM. In case of unfavorable anatomy, however, accurate preoperative planning is mandatory to avoid technical failure and systemic complications.

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