True superior gluteal artery aneurysm

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Gluteal artery aneurysms (GAAs), classified as true or false, account for <1% of all aneurysms. Of the 175 GAAs reported in the English literature, 20 are true aneurysms, making this case the twenty-first reported true aneurysm and sixth of atherosclerotic origin. We report a true GAA in a 72-year-old woman. Pelvic computed tomography suggested GAA (7.2 × 4.9 cm); subsequently, an endovascular approach allowed definitive diagnosis and treatment—coil embolization. In accordance with the literature and the surgical and clinical success of our case, endovascular embolization of GAAs has emerged as an effective and safe treatment. (J Vasc Surg Cases 2015;1:221-3.)

An aneurysm—meaning "dilation"—is a localized, blood-filled sac in the wall of a blood vessel,[1] broadly classified as a true aneurysm or a false aneurysm (pseudoaneurysm).[2] A gluteal artery aneurysm (GAA) arises from the posterior branch of the internal iliac artery[3] and represents an uncommon clinical finding as well as a unique surgical challenge.[2]

The true incidence of GAAs is unknown, but they are suspected to account for <1% of all aneurysms.[4] Documentation of GAAs in the literature dates back to the 1800s, with the first true GAA being noted in 1973 with an etiology of polyarteritis nodosa.[5] The most recent review of GAAs was in 1995, with a reported 122 GAAs in the English-language literature.[6] Since 1995, 53 GAAs have been reported to date. Of the 175 GAAs reported, only 20 are true aneurysms, making this case the twenty-first reported true aneurysm and sixth of atherosclerotic origin. The case and images are reported with the consent of the patient.

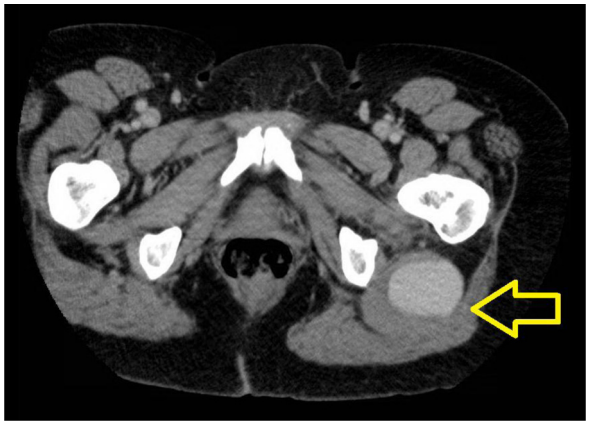

Fig 1. Pelvic computed tomography: axial image demonstrating large, nonhomogeneously enhanced 7.2- × 4.9-cm deep left gluteal mass structure, suggesting aneurysm of gluteal branch of left internal iliac artery.

CASE PRESENTATION

A 72-year-old African American woman presented to the emergency department complaining of left gluteal pain, without etiopathogenesis of trauma or infection, of 3 months in duration. The patient described the pain as sharp and progressive, with radiation down the posterior left thigh to the level of the knee. The patient was a nonsmoker without record of antiplatelet or anticoagulant therapy and with a past medical history of peripheral vascular disease, hypertension, and hyperlipidemia.

Physical examination of the left lower gluteal region revealed a tender and pulsating mass. All peripheral pulses were present and equal with no neurologic deficits. Vital signs, cell counts, and blood chemistries were within normal limits. The suspicion of aneurysm was confirmed with pelvic computed tomography demonstrating a nonhomogeneously enhanced 7.2- × 4.9-cm deep left gluteal mass, suggesting an aneurysm of the gluteal branch of the left internal iliac artery (Fig 1).

After the technique, risks, and benefits of open and endovascular approaches were explained, the patient decided to undergo an endovascular aneurysm repair. In the vascular suite, transfemoral angiography was performed using the up-and-over technique (75 mL of iodixanol contrast agent, dose-area product of 25 mGy-cm^2). Angiography showed normal aortoiliac anatomy. Selective angiography of the left internal iliac artery revealed a solitary saccular aneurysm of the superior gluteal artery without extravasation of contrast material (Fig 2, *A*). Superselective embolization was achieved by deployment of 32 platinum coils (Tornado and Nester coils; Cook Medical, Bloomington, Ind) within the aneurysm itself and at the outflow and inflow tract (Fig 2, *B*). A completion angiogram revealed discontinuation of outflow from the superior gluteal artery and the aneurysm sac, no extravasation, and no retrograde filling (Fig 2, *C*).

The patient was discharged on the first postoperative day, with complete resolution of gluteal pain and pulsation and instructions

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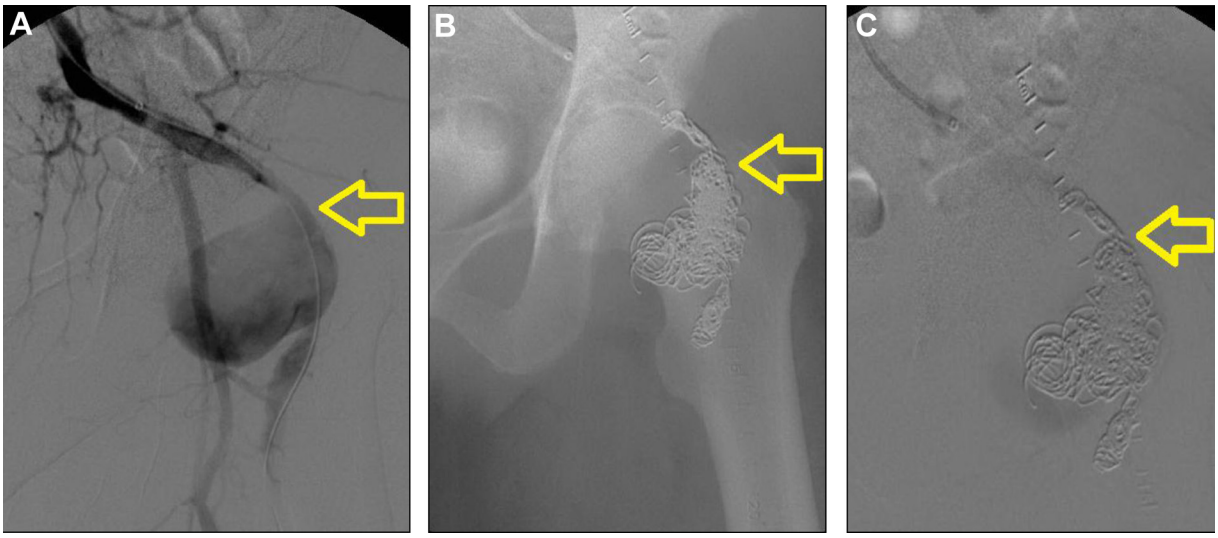

Fig 2. A, Pre-embolization angiography: left internal iliac artery with saccular aneurysm of superior gluteal artery. **B,** Superselective embolization of aneurysm: deployment of coils within the aneurysm at the outflow and inflow tract. **C,** Completion angiography: discontinuation of outflow from superior gluteal artery and the aneurysm sac, no extravasation, and no retrograde filling.

for resumption of normal daily activities. Follow-up at 3 months and 3 years revealed no recurrence of clinical symptoms; therefore, no further imaging was indicated.

DISCUSSION

Anatomy. The gluteal arteries arise from the posterior branch of the internal iliac arteries and are separated by the piriformis muscle as they exit through the greater sciatic foramen. These vessels supply the gluteal muscles and accompany the superior and inferior gluteal and sciatic nerves.[3,7] Because of the anatomy, disorders of these vessels typically are manifested with associated symptoms of mass effect.[8]

Pathogenesis. Aneurysms can broadly be divided into two classes: true aneurysm, expansion of all three layers of the vessel wall (intima, media, adventitia); and false aneurysm (pseudoaneurysm), disruption of one or more layers of the vessel wall as a consequence of injury, with collection of blood confined by the surrounding tissue.[9] Therefore, according to definition, one can distinguish between a true aneurysm and a false aneurysm through a detailed history. Most GAAs are pseudoaneurysms, with a mechanism of trauma; leading causes are pelvic fracture, perforating injury, and iatrogenic origin.[10] True aneurysms are extremely rare, with reported causes being mycotic,[2,11-16] atherosclerosis,[4,6,10,17,18] polyarteritis nodosa,[5] arteriovenous malformation,[19] and intimal-medial mucoid degeneration.[20] The most common etiology of true aneurysms is mycotic. The endovascular approach does not allow histopathologic confirmation of etiology; hence, the etiology of endovascular cases was categorized on the basis of history, physical examination findings, and morphologic features of the aneurysms. One can further classify GAAs into inferior and superior branches, the latter being threefold more common.[6,14] Left is more common than right, with bilateral aneurysms being reported once in 2009, and men are more commonly affected than women are.[15]

Diagnosis. Presentation of a true GAA usually has no history of trauma with an associated pulsating mass.[2] Compression of nerves may be present with complaints of sciatica, insensate pudendal nerve, and regional pain and muscle weakness.[6] Differential diagnosis should include the following: hematoma, abscess, enlarged bursa, granulomatous disease, arteriovenous malformation, hygroma, lipoma, neoplastic disease, sciatic hernia, and echinococcal cysts.[7] Investigation must reveal a connection with the vascular system. Diagnostic tools include duplex Doppler ultrasonography, computed tomography, magnetic resonance imaging, and angiography.[2,16] Angiography, being the "gold standard," can confirm diagnosis by demonstrating connection with the vasculature and help direct treatment from mapping of the distal circulation.[7,17]

Surgical strategy. Treatment of GAAs is recommended when the diameter is >25 mm, when they are symptomatic, and if there is risk of rupture or injury to the sciatic nerve.[4] Because of the possible morbidity (exsanguinating hemorrhage, distal embolism, thrombosis) associated with this aneurysm,[15,20] it is recommended to manage it aggressively with implementation of surgical therapy soon after diagnosis.[13] Treatment involves ligation or occlusion, through an open or endovascular approach. Since 1898, standard treatment options involve a two-stage operation through a transperitoneal or retroperitoneal approach for inflow control to the aneurysm by internal artery compression and combined with direct aneurysmorrhaphy.[6] Several modifications of this technique have since been reported, and the open technique remains the basis of the traditional therapeutic modality.[6,11]

The open surgical approach is effective; however, it is not an innocuous operation, carrying a high risk of intraoperative bleeding, muscle necrosis, and injury of adjacent structures.[16] Therefore, an alternative of endovascular technique has effectively been developed with a decrease of mortality and morbidity associated with GAA repair.[12,17] The endovascular approach prevents the need to enter the retroperitoneal space, with resulting decreased risk of infection and iatrogenic nerve and vasculature injuries.[17] The effectiveness of the endovascular approach is due to elimination of arterial pressure and post-thrombotic tissue remodeling; therefore, timing of symptom resolution may not be immediate as expected in an open approach.[12] Endovascular modalities of transcatheter embolization include the following: hemostatic agents, such as thrombin injection, Gelfoam (Pfizer, New York, NY), and Floseal (Baxter International Inc, Deerfield, Ill); coils; stent-graft placement; thrombosis by 48-hour balloon occlusion; sclerotherapy; and vascular plugs.[2,16-18] This particular case used transcatheter coil embolization. From 1977 to 1995, transcatheter embolization was tried in 7 of 19 documented cases[6]; however, from 1995 to 2015, transcatheter embolization was the definitive treatment in 44 of 53 GAA cases.[2,4,7,15-20]

CONCLUSIONS

A true GAA is a rare entity without conventional etiopathogenesis; however, because of possible associated morbidity and mortality, it is recommended to manage it aggressively with implementation of surgical therapy soon after diagnosis.[11,13,20] The open surgical approach is the traditional treatment of GAAs, with a recent shift toward the endovascular approach.[18] In accordance with the noted literature and the surgical and clinical success of our case, endovascular embolization of GAAs has emerged as an effective and safe treatment.

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