Gluteal ischemic gangrene due to chronic aortoiliac occlusive disease

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ABSTRACT

Gluteal gangrene rarely complicates interventional procedures such as angiographic embolization in the pelvis or may develop postoperatively after open ligation or endovascular covering or embolization of one or mainly both internal iliac arteries in abdominal aortic surgery if collateral circulation is compromised. On the other hand, gluteal gangrene as a primary manifestation of chronic aortoiliac occlusive disease is very exceptional in literature. We present a patient with atherosclerotic aortoiliac obstruction and a necrotic eschar on her left buttock treated with aortobiiliac bypass after digital subtraction angiography. Internal iliac artery revascularization, even contralaterally, is crucial for the healing of necrotic tissue in these patients. (J Vasc Surg Cases Innov Tech 2025;11:101792.)

Keywords: Leriche syndrome; Peripheral arterial disease; Buttock; Gluteal; Gangrene

Aortoiliac occlusive disease (AIOD) is a substantial form of peripheral arterial disease that is defined by the obstruction of the infrarenal aorta and/or the common iliac arteries (CIAs). Claudication, erectile dysfunction, and diminished distal pulses are the main findings (also known as Leriche triad).[1-3] AIOD afflicts approximately 12% to 20% of people over 65 years.[2] Cigarette smoking, dyslipidemia, male gender, diabetes mellitus, and hypertension are the main predisposing factors.[4] Progression to foot gangrene usually requires additional pathology at the femoral and/or the tibial level.[2] Gluteal gangrene resulting from AIOD is scarce in the literature. It may complicate angiographic embolization for retroperitoneal bleeding in pelvic trauma.[5,6] Moreover, it may result after open ligation or endovascular covering or embolization of one or mainly both internal iliac arteries (IIAs) in abdominal aortic surgery if collateral circulation is compromised.[5-7] We present an exceptional case of buttock ischemia resulting in gluteal gangrene as a complication of severe chronic AIOD. The patient agreed to publish their case details and images. Informed consent has been obtained. This case report was approved by the institutional review board of the University Hospital of Patras (IRB no. 29/11-07-2018).

CASE PRESENTATION

A 52-year-old woman presented with early onset local skin gangrene at her left buttock and intermittent claudication at a 50 m distance during the prior 6 months. She was an active smoker and overweight (body mass index: 28); her past medical history included arterial hypertension, dyslipidemia, and hyperthyroidism, and she was negative for vasculitis or autoimmune disease. She was receiving rosuvastatin 20 mg once daily (o.d.), nifedipine 30 mg o.d., and thiamazole 5 mg o.d. Peripheral pulses were not palpable, and the ankle-brachial index was 0.4 bilaterally. Stiffness was realized under the left buttock necrotic eschar on palpation, demonstrating a deeper extent of tissue necrosis (Fig 1). She had already undergone an abdominal computed tomography, which revealed obstruction of the distal aorta and CIAs. She denied admission to the hospital and left under clopidogrel 75 mg o.d. and advised to perform a diagnostic workup as an outpatient. She came back after a month with an ulcer at her left buttock after a needle biopsy performed by an orthopedic surgeon. He tried to exclude malignancy as a suspicious mass was depicted under the necrotic skin in magnetic resonance imaging. Biopsy revealed ischemic and necrotic tissue lesions. After admission, she underwent a digital subtraction angiography, which revealed obstruction of the distal aorta and CIAs. The right IIA was patent but the left was obstructed. A rich collateral network was apparent (Figs 2 and 3). Under local care, satisfactory healing was in process. The patient denied open aortic reconstructive surgery and was discharged. No endovascular option was offered due to the better long-term patency of open repair in this relatively young patient. After 2 months, she came back with a large ischemic wound at her left buttock. During this interval, she received hyperbaric oxygen at another institute. She was admitted and underwent an aortobiiliac bypass with a bifurcated synthetic 16 mm × 8 mm polytetrafluoroethylene graft (W. L. Gore & Associates Inc). All anastomoses were end to side. The right distal anastomosis was performed at iliac bifurcation and the left at the distal

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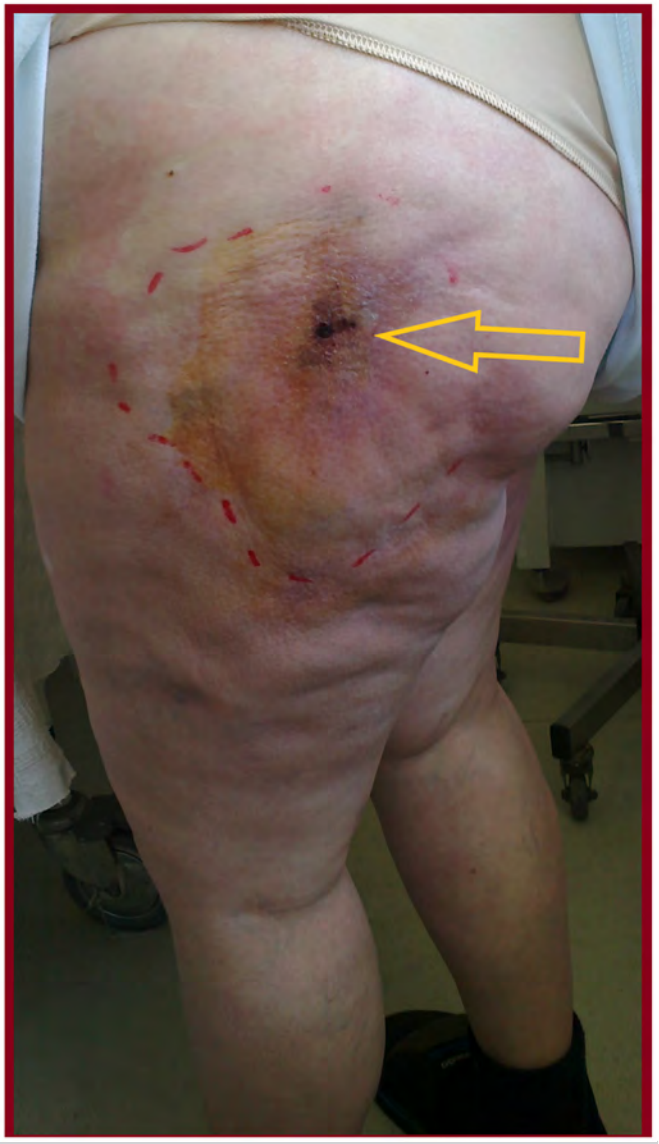

Fig 1. Ischemic area with a reddish appearance on the left buttock and a necrotic eschar within (*orange arrow*).

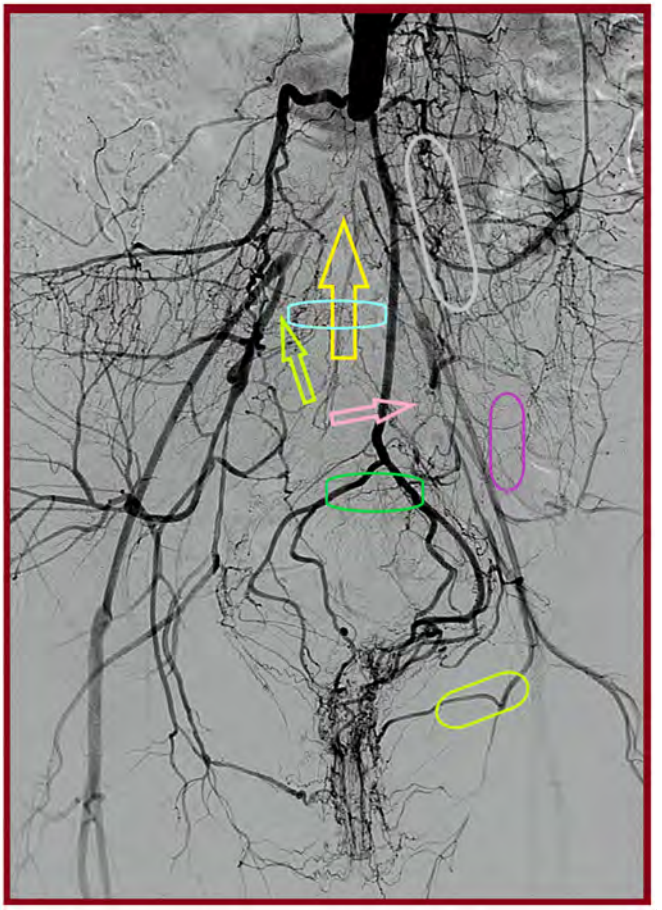

Fig 2. Obstructed aortoiliac bifurcation (*yellow arrow*). The right internal iliac artery is patent (*green arrow*), whereas the left is obstructed (*pink arrow*). A rich collateral network is apparent (*gray area*: anastomotic branches with the lumbar/iliolumbar artery, *light blue area*: sacral arteries, *purple*: deep iliac circumflex artery anastomosed with the subcostal artery, *green area*: inferior mesenteric artery anastomosed with the rectal artery, *light green area*: external iliac artery anastomosed with the inferior epigastric artery, also known as the Winslow pathway).

external iliac artery. No jump graft to the left distal IIA was performed as external iliac artery retrograde flow would supply the distal IIA branches through the collateral network. Postoperatively, distal pulses were palpable. The wound was healed 6 months after debridement and local care with wound dressings. She remained asymptomatic for the following 12 years when came back for an asymptomatic carotid artery stenosis, but she denied endarterectomy (Fig 4). She was obese (body mass index: 39). Ankle-brachial indices were 0.9 bilaterally. She has not smoked since the operation. Computed tomography angiography confirmed normal graft patency (Fig 5).

DISCUSSION

Buttock claudication (BC) is a classic symptom in 10% to 60% of patients with chronic AIOD. BC development depends on the extent of arterial occlusions and the amount of collateral supply.[8] On the other hand, buttock ischemic skin lesions or necrosis of the deeper gluteal muscle complicating chronic AIOD is extremely rare. It is caused by the obstruction of one or both IIAs or by the absence of inline flow to patent IIAs due to proximal AIOD. By the progression of the disease, BC may lead to buttock ischemic lesions if collateral circulation does not efficiently compensate for the diminishing blood supply.[8] Local pressure due to supine position (during sleeping), obesity, and IIA disease are synergistic factors for buttock necrotic wounds. Atypical ischemic ulcerations in the form of macules, purpura, nodules, and livedo reticularis may develop in the proximal leg, including the buttocks, even in the presence of palpable distal pulses, although most patients have a borderline ankle-brachial index, like our patient.[9-11]

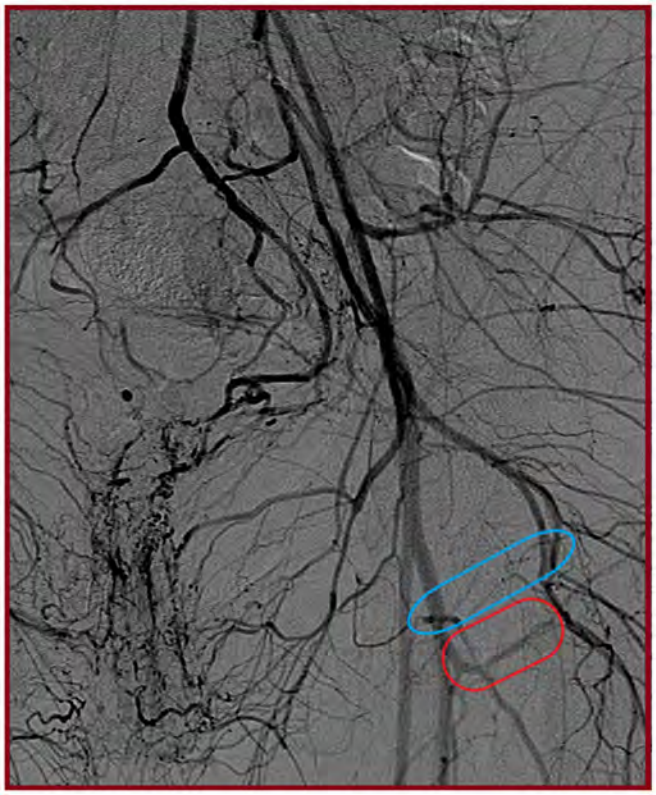

Fig 3. Additional collateral network at the left buttock (*blue area*: medial femoral circumflex artery anastomosed with the superior gluteal artery, *red area*: lateral femoral circumflex artery anastomosed with the inferior gluteal artery).

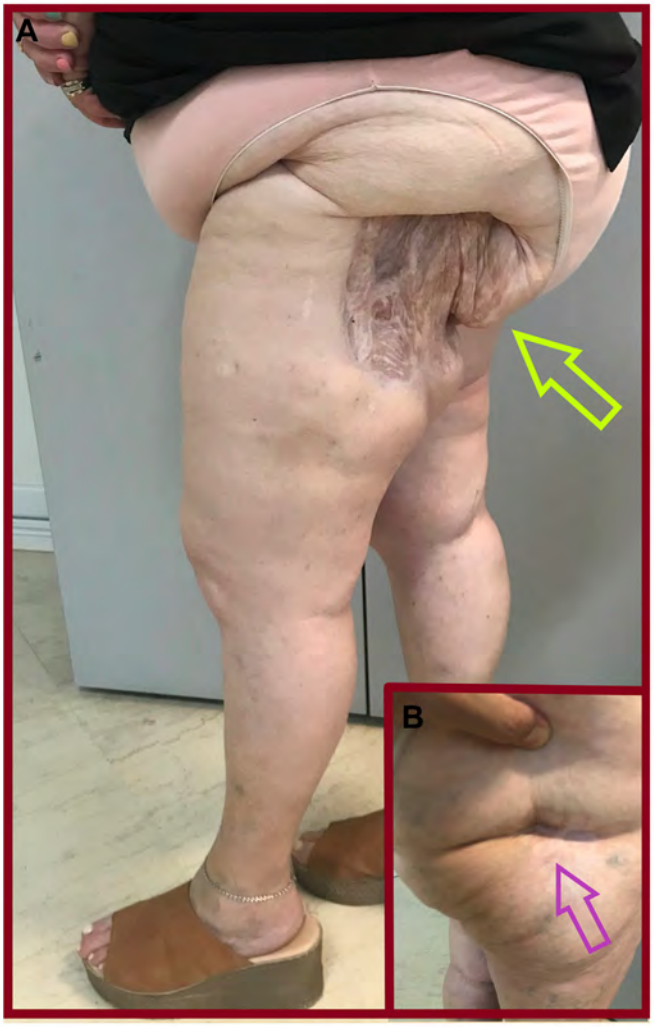

Fig 4. The appearance of the buttocks after 12 years. **A,** The muscle deficit of the left buttock (*green arrow*). **B,** The skin mark on the right buttock indicative of the previous ischemic episode (*purple arrow*).

We were not able to reveal a similar case of a de novo gluteal necrosis and chronic AIOD in the literature. Most reported cases have a history of antecedent vascular procedures, as bypass surgery and angioplasty (or stenting) in AIOD may fail to revascularize efficiently the IIA territories, making these patients susceptible to pelvic and buttock ischemia. Exertional BC in 28% of patients after aortobifemoral bypass was detected by Jaquinandi et al in 2007.[12] An interesting case was reported by Kemmochi et al.[13] Buttock ischemic skin lesions were attributed to ligation of the right IIA during aortoiliac bypass for aortoiliac (including IIA) aneurysms, whereas the left IIA was occluded. It was treated with a redo operation including a bypass to the right IIA and a lumbar artery.[13] In another case reported by Coruh et al,[14] insufficient pelvic perfusion 5 months after aortobifemoral bypass predisposed to gluteal necrosis. An additional case was reported by Simman et al,[15] where bilateral ischemic buttock necrotic wounds were treated with debridements and vacuum-assisted closure as the patient was not fit for additional revascularization options. This case highlights the rule that "any necrotic wound at the buttocks should not be classified as pressure wound before clinical examination of the vascular status has been made."[15] At last, one reported case by Paone et al,[4] gluteal necrosis was in the context of acute ischemia (acute on chronic aortoiliac occlusion) and not chronic AIOD. In such cases, urgent revascularization is essential to prevent necrosis as collateral circulation is usually not efficient.

The crucial role of IIAs in buttock ischemia is emphasized in the literature. In an interesting case series of 10 patients, isolated IIA occlusive disease was the cause of BC without symptoms in distal legs in most of them.[11] Direct IIA revascularization solved the problem in 85% of them with a 72.5% patency rate in 5 years. It is noteworthy that 20% of them had preoperatively isolated proximal ischemia without distal ischemia.[8] The pelvis deserves an extensive arterial network that dilates and expands in AIOD. The superior gluteal artery and the inferior gluteal artery arise from the IIA and provide blood to the gluteal muscles.[16] The IIA collaterals are the

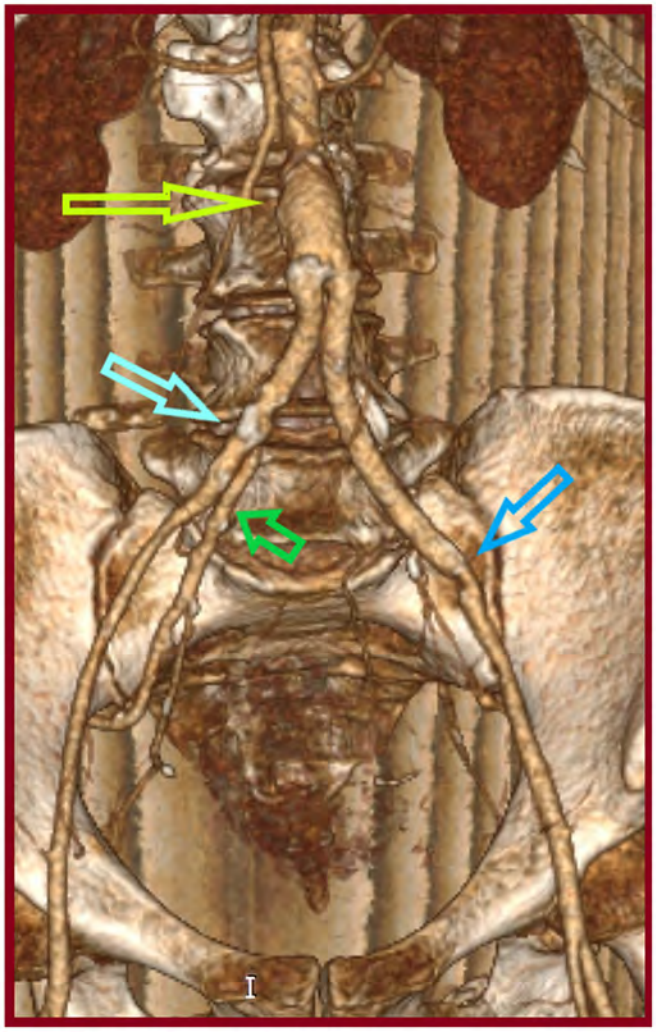

Fig 5. Computed tomography angiography confirmed normal patency of the aortoiliac graft 12 years later (proximal anastomosis: *light green arrow*, right distal anastomosis: *light blue arrow*, left distal anastomosis: *blue arrow*, patent right internal iliac artery: *green arrow*).

iliolumbar artery, superior gluteal artery, inferior gluteal artery, internal pudendal artery, obturator artery, and the central anastomotic system between visceral (umbilical, middle rectal, and genitourinary) and parietal (superior and inferior sacral) arteries.[16,17] In addition, in AIOD, significant collaterals may develop between the branches of both IIAs and the deep femoral artery branches. This explains why the obstruction of one or both IIA can remain asymptomatic in some patients.[8,18] In our patient, revascularization with direct flow in the right IIA restored the ischemia in the left buttock through collateralization between IIAs and additional blood supply from the left profunda femoris artery branches (Figs 2 and 3).[8]

Ischemic fasciitis, malignancy, vasculitis, Takayasu's arteritis, and thromboangiitis obliterans may rarely be considered.[4,14,19,20] After revascularization, surgical debridement and local wound care with dressings and vacuum-assisted closure are very important to heal large wounds.[15]

CONCLUSIONS

Gluteal necrotic eschars need a vascular consultation to exclude severe pelvic ischemia even in the absence of signs and symptoms distally. Imaging studies are essential to diagnose AIOD and/or IIA stenosis or obstruction. Prompt revascularization and care should lead to a good outcome.

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DISCLOSURES

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