An internal iliac artery aneurysm causing sudden buttock ischemia and nerve root compression

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Acute buttock ischemia can be a consequence of aneurysmatic disease and has a dramatic presentation. This case report describes an otherwise healthy patient with a simultaneous onset of buttock ischemia combined with sciatic nerve compression caused by a small distal internal iliac artery aneurysm. Coiling of the aneurysm prevented thromboembolism recurrence but was only partially successful in reducing the symptoms of nerve compression. Given the serious consequences, prophylactic treatment independent of aneurysm diameter can be considered. (J Vasc Surg Cases 2015;1:151-3.)

Isolated internal iliac artery aneurysms are rare and mostly without symptoms until compression of adjacent structures or rupture occurs. Treatment recommendations are based on diameters at risk of rupture. We describe a patient with an initially unrecognized case of coinciding buttock ischemia and nerve compression caused by an iliac artery aneurysm that would otherwise not have met treatment criteria. Consent was obtained from the patient to publish these images and medical history.

CASE REPORT

On the evening of Christmas Day 2013, a 77-year-old man presented at our emergency department. Apart from gastric ulcer surgery 40 years earlier, the patient had an unremarkable medical history. He called on his general practitioner 3 days before because of a sudden start of pain isolated in his right buttock with purple stains and a coinciding onset of a numb feeling and a weight-bearing inability of his right lower leg and foot. There was no history of trauma or claudication. He was initially diagnosed with herpes zoster, but when the pain became intolerable he was referred to our hospital.

Examination showed a vital patient with purple/blue skin coloration of his right buttock and severe pain when local pressure was applied (Fig 1). Clear pulsations could be felt of the common femoral and popliteal artery, and Doppler signals of the dorsal pedis and posterior tibial artery were normal. The ankle-brachial pressure index was 0.94, and capillary refill of his foot was normal. Neurologic examination showed a decrease in sensibility from his knee down, most prominent on the lateral side, and complete loss of sensibility of his foot and toes. Impairment of active knee and plantar foot flexion, combined with absent dorsal flexion, suggested sciatic nerve involvement.

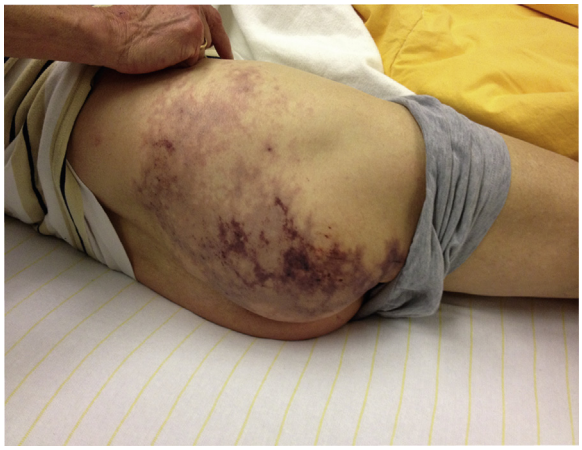

Fig 1. Ischemic right buttock on presentation.

Results of blood tests were within normal reference ranges apart from a mild leucocytosis (11×10^9/L) and elevated creatine kinase (10,155 U/L), reflecting muscle tissue loss. Magnetic resonance imaging of the lumbar spine ruled out vertebral disc protrusion. A computed tomography angiogram of the thoracoabdominal aorta showed an intact aneurysm of the distal internal iliac artery, the superior gluteal artery with a diameter of 25 mm, and extensive intraluminal thrombus (Fig 2, *A*). No other aortoiliac aneurysm was seen.

Our patient was heparinized, and diuresis was forced to prevent myoglobulinemia-induced kidney damage. The next morning, the aneurysm was coiled (Fig 2, *B* and *C*), and the therapeutic dosed heparin was discontinued. Blood levels of creatine kinase normalized in 7 days. The pain gradually subsided, and the buttock wounds healed. The motor and sensory loss also diminished, although impairment of active dorsal flection of the foot persisted.

Magnetic resonance imaging performed 6 weeks after coiling showed compression of the L5 and S1 roots of the sciatic nerve by the aneurysm (Fig 2, *D*). Four months after onset, the dorsal flection impairment remained, and he developed a hyperemic right lower leg and foot reflecting compression-induced sympathetic nerve inhibition.

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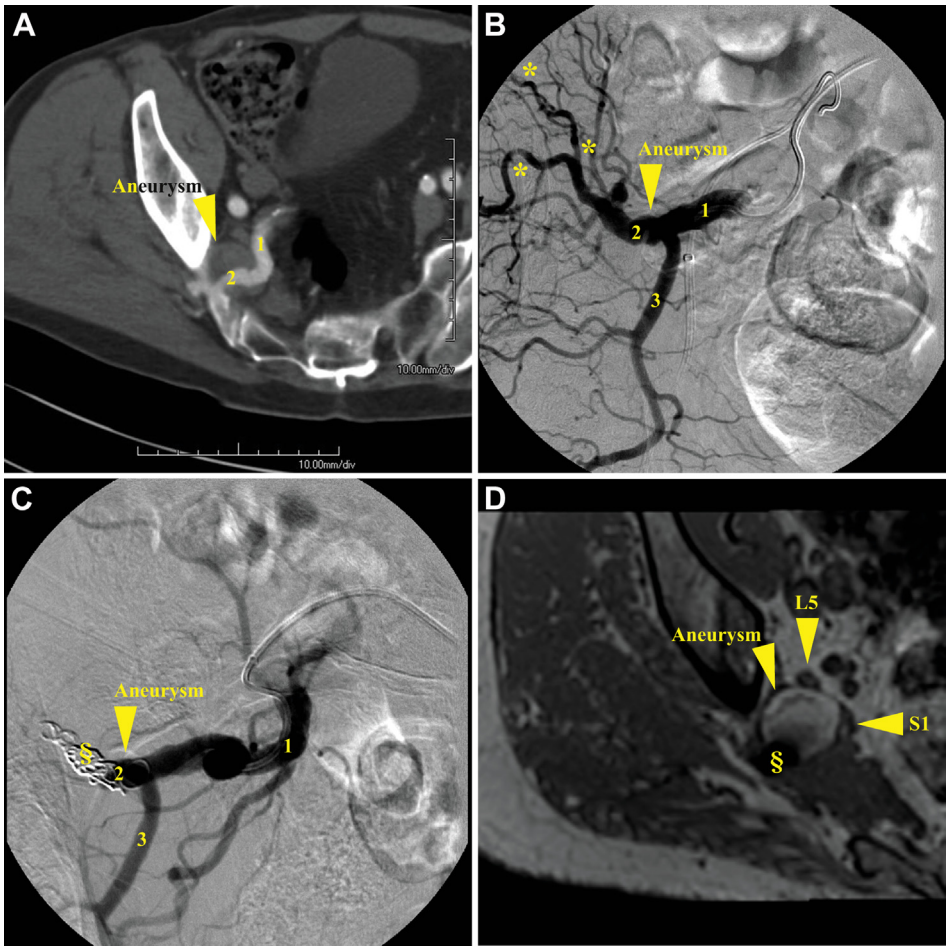

Fig 2. A, Computed tomography angiogram shows the internal iliac artery (*1*) aneurysm (*arrowhead*) with intraluminal thrombus and the superior gluteal artery (*2*). **B,** Angiogram shows the internal iliac artery (*1*) and the superior (*2*) and inferior (*3*) gluteal artery. Embolic thrombus (*) can be seen distally from the aneurysm (*arrowhead*). **C,** Angiogram after coiling (§) of the superior gluteal artery. Because the aneurysm (*arrowhead*) is difficult to distinguish angiographically, the aneurysm was not completely coiled; however, outflow was obstructed successfully. **D,** Transverse T1-weighted image shows the close relationship of L5 and S1 nerve root to the aneurysm. Note the absent flow void in the aneurysm after coiling (§).

DISCUSSION

Isolated internal iliac artery aneurysms occur with an incidence of 0.4% and are mainly caused by progressive atherosclerosis.[1] Although relatively rare, these aneurysms are often asymptomatic until manifestation with rupture in 40% of patients, with a 31% death rate.[2] Rupture risk-based treatment has been recommended for aneurysms ≥30 mm in diameter.[2]

Possible symptoms before rupture include compression of the adjacent structures, including the ureter, iliac veins, lumbosacral plexus, obturator nerve, colon, and small bowel.[2-4] Thromboembolic complications potentially occur analogous with other types of arterial aneurysms but have not been described before. In this patient, emboli caused ischemia of the right buttock, and clinically the S1 and L5 roots of the sciatic nerve were affected.

Thromboembolic nerve ischemia and nerve compression were considered as possible explanations. Georgakis et al[5] studied the arterial supply to the sciatic nerve and described sciatic arteries mainly originating from the medial circumflex femoral artery and the inferior gluteal artery, but not from the superior gluteal artery. Given the location of the aneurysm in the proximal superior gluteal artery (Fig 3), thromboembolic nerve ischemia was considered unlikely. The simultaneous onset of symptoms was explained by compression of the sciatic nerve roots, most likely due to increased radial forces in the aneurysm caused by the partial outflow obstruction. Open surgical treatment and ligating or resecting the aneurysm was considered too risky given the very distal location of the aneurysm. Coiling of the aneurysm prevented recurrence of thromboembolic symptoms but only partially reduced nerve compression signs.

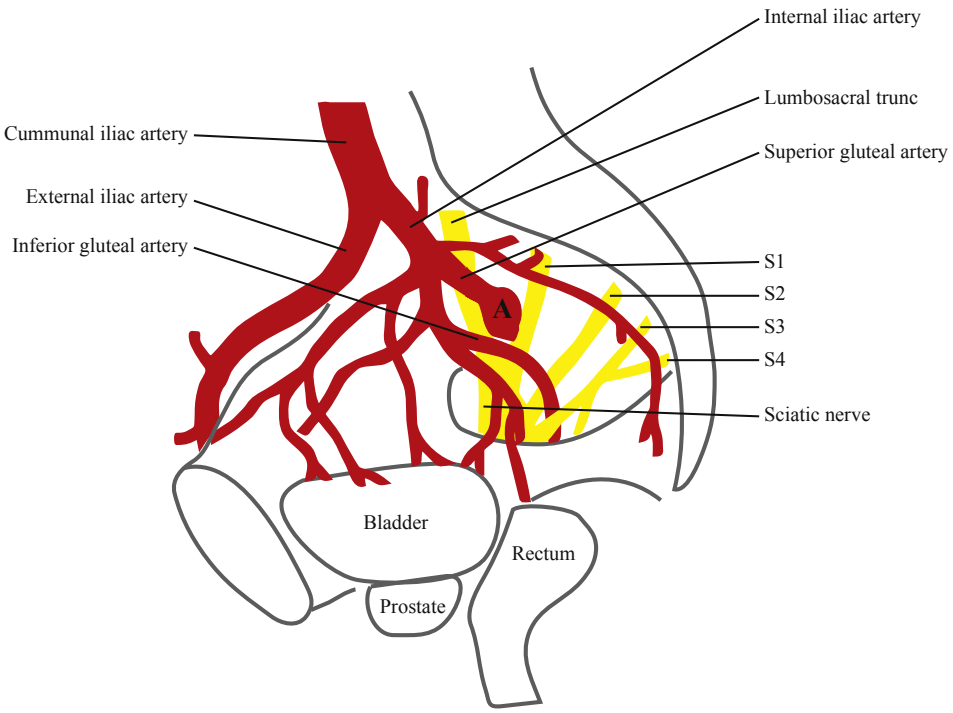

Fig 3. A schematic overview shows the anatomy and position of the aneurysm (*A*).

CONCLUSIONS

Our case demonstrates the multidimensional presentation, risk of nerve compression, and thromboembolisms, which could advocate early treatment independent of diameter.

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