

# Persistent sciatic artery presenting with limb ischemia

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The persistent sciatic artery (PSA) is a rare but clinically significant congenital vascular anomaly. Clinical presentation varies and PSA can cause a number of complications, including limb loss. We describe the presenting features and treatments in two patients. The former was found to have thrombosis of a PSA with distal thromboemboli and was treated with a bypass graft. The latter was treated for an ischemic foot following successful ruptured aortic aneurysm repair and was found incidentally to have patent PSA with concomitant stenosis of the common iliac artery, which was successfully treated with stent grafting. (*J Vasc Surg* 2013;57:225-9.)

In 1832, Green first described the persistent sciatic artery (PSA) as a variant of the femoral artery and highlighted “the utility of speedily making known to the profession those irregularities in the arterial system, which are the more dangerous to the operator as they are rare and unnoticed in our popular works.”<sup>1</sup> Since Green’s description, more than 160 cases have been reported,<sup>2</sup> and several authors have reviewed the body of PSA literature in the last few years.<sup>3,4</sup>

PSA is a relatively rare anomaly and can have highly variable clinical presentation. Nevertheless, the importance of PSA cannot be overstated, as a missed diagnosis may lead to disability or limb loss.<sup>5</sup> We have had the opportunity to treat and present herein patients with two different clinical presentations.

## CASE REPORTS

**Case 1.** A 41-year-old male presented with a 2-week history of right calf claudication and foot tingling with reduced walking distance (50 meters). His history was significant for diabetes mellitus and dyslipidemia but no hypertension, cardiac symptoms, or previous thromboembolic events. Physical examination of the right leg revealed palpable femoral pulse, faint popliteal pulse, and absence of the posterior tibial and dorsal pedal pulses. Left lower extremity pulses were normal. Arterial duplex scanning demonstrated occlusion of the right popliteal artery.

The patient underwent diagnostic angiography. The right common iliac artery, external iliac artery, and common femoral artery were normal. The superficial femoral artery terminated in multiple branches at the level of the adductor canal (Fig 1, A). A hypertrophied deep femoral artery provided a large collateral, which opacified the popliteal artery at the level of the knee (Fig 1,

B). Multiple filling defects were found within the above-knee popliteal artery, and there was total occlusion of the below-knee popliteal artery and tibioperoneal trunk. There were collaterals reconstituting the anterior tibial and posterior tibial arteries. This anatomy was highly suggestive of persistent sciatic artery. Selective arteriography showed the right internal iliac to be unusually large in caliber and occluded abruptly beyond the level of the inferior gluteal artery (Fig 1, C), further reinforcing the diagnosis of PSA. Left lower extremity angiography was unremarkable.

A reversed saphenous vein bypass graft was placed from the right proximal superficial femoral artery to the tibioperoneal trunk and proximal posterior tibial artery after local thrombectomy of the tibioperoneal trunk. Completion arteriogram demonstrated prograde flow into the peroneal and proximal posterior tibial arteries with retrograde flow into the tibioperoneal trunk and into the anterior tibial artery. On postoperative follow-up at 6 and 12 months, the patient complained only of slight numbness near the right ankle but was otherwise asymptomatic. Right popliteal and posterior tibial pulses were palpable, the ankle-brachial index was normal, and arterial duplex scanning demonstrated a patent bypass graft at each of those postoperative visits.

**Case 2.** A 73-year-old female presented with a 3-day history of progressively worsening left foot pain at rest. This pain was worse on walking and was associated with her toes turning blue. Her history was significant for hypertension, dyslipidemia, and open repair of a ruptured abdominal aortic aneurysm 14 days prior to our initial evaluation. The ruptured aneurysm was repaired with an 18-mm diameter Hemashield polyester tube graft (Maquet Cardiovascular, Wayne, NJ). Intraoperatively, the right femoral pulse was lost, which was restored with an 8-mm-diameter Gore-Tex ringed jump graft (W. L. Gore, Flagstaff, Ariz) to the right common iliac artery. Physical examination of the left leg revealed absent femoral, popliteal, posterior tibial, and dorsal pedal pulses with cyanotic appearance of her left foot and an ankle pressure of 50 mm Hg. Right femoral, popliteal, and dorsal pedal pulses were palpable, and the right foot appeared well-perfused.

The patient underwent diagnostic angiography. The infrarenal aortic graft was patent, but the left common iliac artery had severe stenosis at its origin (Fig 2, A). The left external iliac, common femoral, deep femoral, and superficial femoral arteries were each diffusely small in caliber. However, a large left-sided PSA was found that continued through the thigh to supply the popliteal

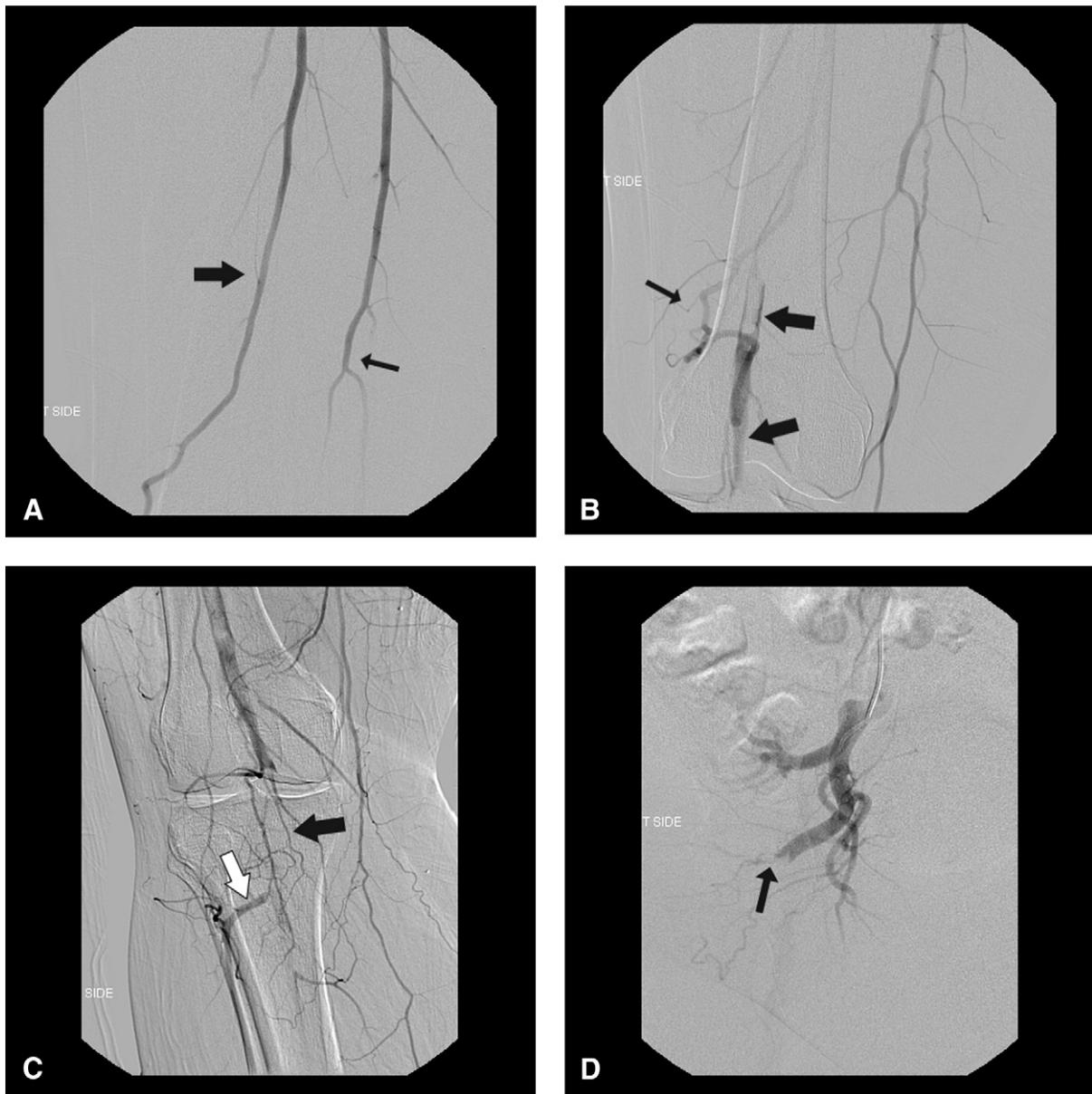
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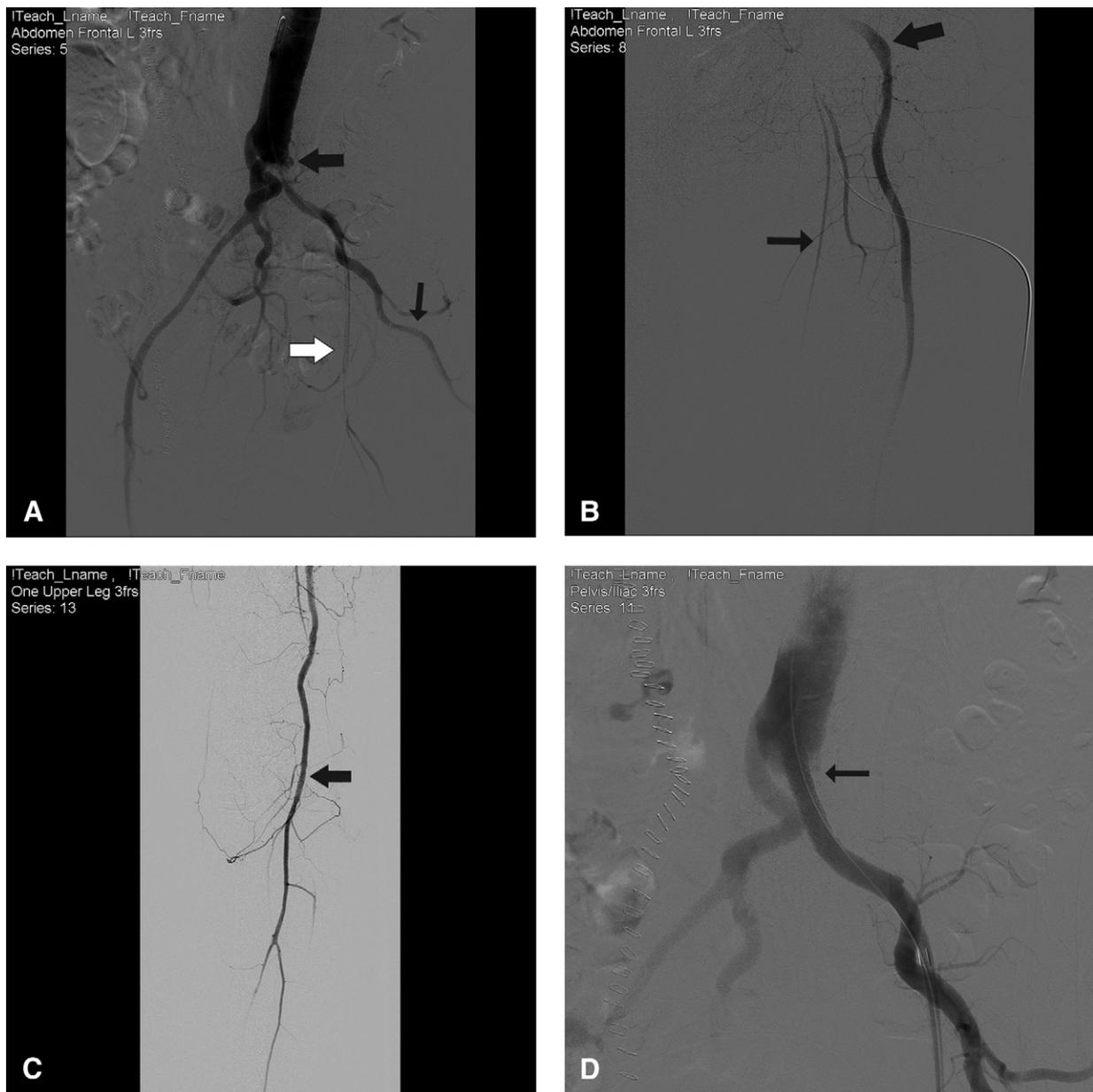


**Fig 1.** Right leg angiography via left transfemoral approach. **A**, The deep femoral artery (*thicker arrow*) continues to join a genicular branch, the superficial femoral artery terminates in the distal thigh (*thinner arrow*). **B**, The deep femoral artery supplies a genicular artery (*thinner black arrow*) that communicates with the popliteal artery containing multiple filling defects, including (**C**) total occlusion of the below-knee popliteal artery (*thicker black arrows*) and tibioperoneal trunk with reconstitution of the anterior tibial artery (*white arrow*). **D**, Selective injection of the internal iliac demonstrates it to be large with an abrupt occlusion (*arrow*) consistent with thrombosis of the persistent sciatic artery.

artery, which demonstrated no anomaly. There was a small fusiform aneurysm of the gluteal portion of the PSA (Fig 2, B), and the PSA reconstituted the popliteal artery (Fig 2, C). Right lower extremity angiography was unremarkable.

The patient's symptoms were attributed to left common iliac artery stenosis and the PSA did not appear to be contributing to her symptoms. A 9-mm-diameter by 38-mm length iCAST stent graft (Atrium, Hudson, NH) was deployed across the stenosis of the left common iliac artery with an excellent technical result

(Fig 2, D). Intervention for the small aneurysm of the PSA would likely have required a bypass from the common iliac artery to some distal target and was not performed given the small size of the aneurysm, very recent ruptured aneurysm repair, lack of apparent distal emboli, and multiple patient comorbidities. Completion arteriogram showed excellent results. The patient tolerated the procedure well with no immediate complications and had excellent left leg pulses and improvement in foot perfusion following the procedure.



**Fig 2.** Aortogram and left leg angiography via left transfemoral approach. **A**, High-grade stenosis at the origin of the left common iliac artery (*thicker black arrow*), the internal iliac artery continues as the persistent sciatic artery (*thinner black arrow*), and the external iliac artery (*white arrow*) is small and occluded by the diagnostic catheter. **B**, The persistent sciatic artery is demonstrated with a small aneurysm in its gluteal portion (*thicker arrow*), the superficial femoral artery (*thinner arrow*) is small and occludes in the thigh, **(C)** the persistent sciatic artery continues as the popliteal artery (*arrow*), and **(D)** good technical result after placement of covered stent in the left common iliac artery (*arrow*).

## DISCUSSION

Embryologically, the sciatic artery originates from the umbilical artery and passes along the dorsal aspect of the developing skeletal mesenchyme to reach the sole of the foot. The sciatic artery serves as the axial blood supply to the developing lower limb bud during embryogenesis. The sciatic artery originates from the umbilical

artery and passes along the dorsal aspect of the developing skeletal mesenchyme to reach the sole of the foot. By 3 months gestation, the femoral artery has fully developed and become functional and the sciatic artery begins to involute. Under normal circumstances, only segments of the sciatic artery persist as the popliteal and peroneal arteries.<sup>6</sup>

**Fig 3.** Persistent sciatic artery (PSA) classification. *Type 1:* complete PSA and normal superficial femoral artery, both providing flow to the popliteal artery. *Type 2a:* complete PSA and incomplete superficial femoral artery that does not reach the popliteal artery. *Type 2b:* complete PSA and absent superficial femoral artery. *Type 3:* incomplete PSA (*upper part persists*) and normal superficial femoral artery. *Type 4:* incomplete PSA (*lower part persists*) and normal superficial femoral artery. *Type 5a:* complete PSA originating from the median (*middle*) sacral artery and incomplete superficial femoral artery. *Type 5b:* complete PSA originating from the median sacral artery and absent superficial femoral artery.

Anatomically, the PSA follows the sciatic nerve through the sciatic foramen, continues through the posterior thigh, and either terminates as muscular branches or anastomoses with the arterial tree at the level of the knee. When the femoral system fails to develop or the axial artery fails to regress, the sciatic artery persists as a continuation of the internal iliac artery. The PSA follows the sciatic nerve through the sciatic foramen, continues through the posterior thigh, and either terminates as muscular branches or anastomoses with the arterial tree at the level of the knee. Van Hooft et al have classified PSA into five types with varying degrees of sciatic and femoral contribution to distal blood flow<sup>7</sup> (Fig 3). Both of our patients were found to have type 2a with PSA supplying the popliteal artery and incomplete superficial femoral artery.

Angiographic studies have estimated the incidence of PSA at only 2.5 to 4.0 per 10,000.<sup>8</sup> Our patients were found to have unilateral PSA, but reports have found bilateral PSAs in 12% to 22% of cases.<sup>9,10</sup> Approximately 40% of cases are asymptomatic and 50% are incidental findings.<sup>11</sup>

Clinically, patients with PSA are asymptomatic during their early years, but at a mean age of 40 to 50 years old, these patients are usually symptomatic, with equal gender incidence. The most common symptom is leg ischemia due to atherosclerosis or thromboembolism. Some reported studies show that 31% to 63% of the PSA cases had lower limb ischemia, with up to 25% manifested as critical limb ischemia.<sup>5</sup> Compared with normally developed iliofemoral arteries, PSA more frequently shows accelerated atherosclerotic changes leading to aneurysms and thromboembolism.<sup>9</sup> Other symp-

toms may include a pulsating mass in the buttock and radicular pain along the L5-S1 nerve roots. Patients may present with symptoms from a ruptured PSA aneurysm as well. On examination, palpable popliteal and distal pulses with absence of the femoral pulse should raise the suspicion of PSA and the definitive diagnostic test is angiography. Aneurysm formation of the gluteal portion of the PSA, found in 44% to 61% of cases, may be complicated by aneurysm thrombosis, aneurysm rupture, distal embolization, and/or local compression of the sciatic nerve.<sup>12</sup> The formation of the aneurysm has been attributed to two factors: reduced elastic elements in the congenitally hypoplastic arterial wall and a relatively exposed anatomic position of this artery in the buttocks, which predisposes it to chronic trauma.<sup>13</sup> The thromboembolic complications of a PSA aneurysm can lead to amputation of the limb for approximately 10% of patients with PSA despite surgery and endovascular treatment.<sup>9</sup> Our first patient may have had an aneurysm that thrombosed leading to ischemia of the leg, but we cannot be certain of this since no cross-sectional imaging was performed. Our second patient had a small 7- to 8-mm-diameter aneurysm, although unrelated to her symptoms. Due to her advanced age and comorbidities, the decision was made to follow the asymptomatic PSA aneurysm by regular clinical assessment and yearly surveillance color duplex ultrasound. If, however, the size of the aneurysm increases to 1.5 times the diameter of the proximal adjacent segment or mural thrombus is found on follow-up serial duplex US, then we will treat the aneurysm.

Management of symptomatic PSA depends on the presence or absence of an aneurysm, anatomy of the sciatic

and iliofemoral system, the patient's symptoms, and concomitant vascular occlusive disease. Management of asymptomatic PSAs without aneurysm is usually observation with continued yearly surveillance with color duplex ultrasound because of the high incidence of aneurysm formation or thrombembolism.<sup>9,14</sup> In cases with sufficient collateral circulation, symptomatic PSA aneurysms can be obliterated with endovascular embolization with coils or plugs if there is sufficient collateral blood supply. In the absence of sufficient collateral flow, surgical ligation in conjunction with surgical bypass may be needed. A stent graft may be utilized to treat the aneurysm.<sup>15-17</sup> However, one has to be cognizant of the fact that the stent graft may become compressed in the sitting position. Resection of the aneurysm is generally reserved for cases of sciatic radiculopathy due to mass effect by the aneurysm. Surgical treatment usually entails femoral-popliteal bypass graft or obturator bypass with exclusion of sciatic artery aneurysm. Resection of the aneurysm is generally reserved for cases of sciatic radiculopathy due to the mass effect by the aneurysm. Yamamoto et al performed a literature search on all reported cases of PSA from 1965 to 2009.<sup>17</sup> They found 29 limbs with PSA that were treated in which there was intermediate to long-term follow-up. They found that the outcomes of the different treatments for lower limb ischemia and aneurysm repair were satisfactory, and the patients were asymptomatic during the 2-month to 10-year follow-up period. Endovascular treatment of a ruptured PSA aneurysm has also been described.<sup>18</sup>

In conclusion, PSA is a rare congenital variant of the lower limb vasculature, which can cause a variety of complications, including limb loss. Clinical presentation and types of PSA can vary as illustrated by the two cases presented above. Accordingly, treatment varies by patient and a variety of diagnostic tools, including angiography, dictate clinical management strategies. To appropriately identify and treat these patients, physicians must broaden their differential diagnoses and be aware of this rare vascular anomaly.

## REFERENCES

1. Green PH. On a new variety of the femoral artery. *Lancet* 1832;17:730-2.
2. Ikezawa T, Naiki K, Moriura S, Ikeda S, Hirai M. Aneurysm of bilateral persistent sciatic arteries with ischemic complications: case report and review of the world literature. *J Vasc Surg* 1994;20:96-103.
3. Shutze WP, Garrett WV, Smith BL. Persistent sciatic artery: collective review and management. *Ann Vasc Surg* 1993;7:303-10.
4. Abullarrage CJ, Crawford RS, Patel VI, Conrad MF. Diagnostic strategies for the persistent sciatic artery. *Vasc Endovascular Surg* 2009;43:485-9.
5. Aziz ME, Yusof NR, Abdullah MS, Yusof AH, Yusof MI. Bilateral persistent sciatic arteries with unilateral complicating aneurysm. *Singapore Med J* 2005;46:426-8.
6. Mandell VS, Jaques PF, Delany DJ, Oberheuer V. Persistent sciatic artery: clinical, embryologic, and angiographic features. *AJR Am J Roentgenol* 1985;144:245-9.
7. van Hooff IM, Zeebregts CJ, van Sterkenburg SM, de Vries WR, Reijnen MM. The persistent sciatic artery. *Eur J Vasc Endovasc Surg* 2009;37:585-91.
8. Santaolalla V, Bernabe MH, Hipola Ulecia JM, De Loyola Agundez Gomez I, Hoyos YG, Otero FJ, et al. Persistent sciatic artery. *Ann Vasc Surg* 2010;24:691.e7-691.10.
9. de Boer MT, Evans JD, Mayor P, Guy AJ. An aneurysm at the back of a thigh: a rare presentation of a congenitally persistent sciatic artery. *Eur J Vasc Endovasc Surg* 2000;19:99-100.
10. Maldini G, Teruya TH, Kamida C, Eklof B. Combined percutaneous endovascular and open surgical approach in the treatment of a persistent sciatic artery aneurysm presenting with acute limb-threatening ischemia—a case report and review of the literature. *Vasc Endovascular Surg* 2002;36:403-8.
11. Jones WS, Patel MR, Mills JS. A case of mistaken identity: persistent sciatic artery stenosis as a cause of critical limb ischemia. *Catheter Cardiovasc Interv* 2011;77:308-12.
12. Kritsch D, Hutter HP, Hirschl M, Katzenschlager R. Persistent sciatic artery: an uncommon cause of intermittent claudication. *Int Angiol* 2006;25:327-9.
13. Williams LR, Flanigan DP, O'Connor RJ, Schuler JJ. Persistent sciatic artery. Clinical aspects and operative management. *Am J Surg* 1983;145:687-93.
14. Brantley SK, Rigdon EE, Raju S. Persistent sciatic artery: embryology, pathology, and treatment. *J Vasc Surg* 1993;18:242-8.
15. Mousa A, Rapp Parker A, Emmett MK, AbuRahma A. Endovascular treatment of symptomatic persistent sciatic artery aneurysm: a case report and review of literature. *Vasc Endovascular Surg* 2010;44:312-4.
16. Verikokos C, Avgerinos ED, Chatziioannou A, Katsargyris A, Klonaris C. Endovascular repair of a persistent sciatic artery aneurysm. *Vascular* 2010;18:162-5.
17. Yamamoto H, Yamamoto F, Ishibashi K, Yamaura G, Shiroto K, Motokawa M, et al. Intermediate and long-term outcomes after treating symptomatic persistent sciatic artery using different techniques. *Ann Vasc Surg* 2011;25:e9-15.
18. Rezayat C, Sambol E, Goldstein L, Broderick SR, Karwowski JK, McKinsey JF, et al. Ruptured persistent sciatic artery aneurysm managed by endovascular embolization. *Ann Vasc Surg* 2010;24:e5-9.

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