Persistent sciatic artery: Embryology, pathology, and treatment

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Purpose: The purpose of this article is to describe the embryologic development and anomalous persistence of the sciatic artery, pathologic changes that may occur in the persistent sciatic artery, and management of complications related to these pathologic changes.
Methods: Two patients with persistence of the sciatic artery treated in the authors' experience are reported. In addition, the computerized data base of the University of Mississippi Medical Center was searched for patients with other lower extremity arterial aneurysms, and their records were reviewed for possible aneurysm of a persistent sciatic artery. A review of the literature was undertaken to determine the embryologic development of the persistent sciatic artery, the pathologic changes that have been observed in the sciatic artery, clinical findings associated with these pathologic changes, and methods of treatment.
Results: Two patients with persistence of the sciatic artery were treated by the authors. No other cases were identified in 43 patients with 66 lower extremity aneurysms treated at our institution. The first patient presented with acute lower extremity ischemia caused by thrombosis of a sciatic artery aneurysm and was treated successfully with intraarterial thrombolytic therapy followed by interposition graft repair. In the second case an incidental unilateral persistent sciatic artery was identified in a patient with bilateral Buerger's disease presenting with digital gangrene of the contralateral extremity in whom below-knee amputation was eventually required. Bilateral tibial artery occlusion was observed on arteriography. Histologic examination of the amputated limb confirmed findings typical of Buerger's disease, and no microemboli were seen.
Discussion: The sciatic artery is a continuation of the internal iliac artery and is the primary blood supply to the lower limb bud during early fetal development. The sciatic artery normally involutes during fetal development, but remnants persist as the popliteal and peroneal arteries after the superficial femoral artery develops and establishes continuity with the popliteal artery. Persistence of the sciatic artery as the major blood supply to the lower extremity in adults is a rare vascular anomaly that may be of surgical significance. Failure to appreciate the persistent sciatic artery as the major inflow into the lower extremity may lead to inappropriate bypass of apparent occlusive disease of the superficial femoral artery. The persistent sciatic artery is also frequently aneurysmal, which may cause critical limb ischemia resulting from thrombosis or embolization of aneurysm thrombus. Options for vascular reconstruction include interposition graft replacement and standard femoropopliteal bypass grafting if the common femoral artery is sufficiently developed to provide adequate inflow. As with other peripheral arterial aneurysms resulting in thrombosis and extensive distal arterial embolization and thrombosis, intraarterial thrombolytic therapy may be useful in selected cases before definitive surgical revascularization. (J VASC SURG 1993;18:242-8.)

Since first described by Green[1] in 1832, more than 60 cases of persistent sciatic artery have been reported.[2-26] The sciatic artery, a continuation of the internal iliac artery into the popliteal-tibial vessels, represents the major blood supply to the lower limb bud in early embryologic development. With development of the femoral arteries, the sciatic artery normally involutes. The incidence of persistent sciatic artery has been estimated to be between 0.01%[7] and 0.05%.[14] Bilateral persistent sciatic artery has been observed in 12% of cases.[12] There is no apparent sexual prevalence.[15,24]

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This rare vascular anomaly is associated with a high incidence of aneurysm formation, with estimates ranging from 15%[14] to 44%.[24] Critical lower extremity ischemia related to thrombosis of the aneurysm or embolization of aneurysm thrombus has been a common method of presentation. In addition, failure to recognize a persistent sciatic artery as the major blood supply to the lower extremity may lead to an incorrect diagnosis of superficial femoral artery occlusive disease and inappropriate surgical revascularization.[2,18] This report describes our recent experience with two cases of persistent sciatic artery, followed by a discussion of the embryologic development, clinical and radiologic findings, potential complications, and therapeutic options.

CASE REPORTS

Case 1. A 59-year-old man awakened with severe constant pain in his right lower extremity and applied a belt tourniquet below the knee. He then consulted his family physician, who removed the tourniquet and noted that the extremity was cold with diminished perfusion and neurologic function.

Significant medical history revealed that the patient had been evaluated 4 years before the present illness because of numbness and weakness in the right lower extremity below the knee. Slightly diminished motor and sensory function in the sciatic nerve distribution was noted on examination at that time, and evaluation procedures, including computed tomographic scan of the head, carotid duplex scan, electrocardiogram, and echocardiogram, revealed no abnormalities. No etiologic basis for the symptoms was established and no therapy was undertaken. It was noted that the patient had also smoked one pack of cigarettes per day for 45 years.

Physical examination revealed normal femoral pulses bilaterally, normal popliteal and pedal pulses in the left lower extremity, and absent popliteal and pedal pulses in the right lower extremity. The right lower extremity was cool to touch and had diminished deep and light sensation, as well as diminished motor function.

Emergency arteriography was performed via left femoral artery catheterization. A persistent right sciatic artery with aneurysmal dilatation and intraluminal thrombus was identified (Fig. 1). The distal right superficial femoral artery was congenitally hypoplastic and did not supply significant flow to the lower extremity (Fig. 2). Tibial vessels were not visualized in the right lower extremity. The left lower extremity vessels appeared to be normal in anatomy and free of significant disease.

Intraarterial thrombolytic infusion throughout a 12-hour period restored patency of the sciatic, popliteal, and many previously occluded distal vessels. An acute anterior compartment syndrome was suspected after reperfusion and a four-compartment calf fasciotomy was done. Follow-up arteriogram revealed patent sciatic, popliteal, and posterior tibial arteries with filling of the anterior tibial and peroneal arteries via collaterals. Repair of the right sciatic artery aneurysm was performed by a posterolateral approach and interposition grafting with a 10 mm ring-reinforced expanded polytetrafluoroethylene graft. A posterior tibial pulse was palpable at the ankle after revascularization, but a partial foot drop persisted. Debridement of some necrotic muscle from the anterior compartment was required, but the remainder of the limb remained viable and functional. Serologic tests for syphilis showed negative results. Follow-up 15 months after repair revealed excellent distal perfusion with an ankle/brachial index of 1.16 on the right and 1.09 on the left. The patient is functioning and ambulating normally with a foot drop splint.

Case 2. A 37-year-old man was referred to us because of pain and dry gangrene of the left great toe. He had no history of diabetes or claudication. He smoked five to six cigars per day. Physical examination revealed dry gangrene of the left great toe with no signs of sepsis. He had normal femoral and popliteal pulses bilaterally but no palpable pedal pulses in either extremity. The Doppler-derived ankle/brachial index was 0.63 on the left and 1.0 on the right.

Arteriography revealed normal abdominal aorta and iliac vessels. A persistent right sciatic artery was noted to be the dominant supply to the right lower extremity. The superficial femoral artery was hypoplastic and did not communicate directly with the popliteal artery (Fig. 3). The left leg revealed normal femoropopliteal arterial anatomy. The distal tibial, peroneal, and pedal arteries demonstrated diffuse occlusive disease bilaterally (Fig. 4). Surgical revascularization was considered impossible because of inadequate pedal outflow. A great toe amputation and a subsequent transmetatarsal amputation failed to heal, and a below-knee amputation was required. Pathologic examination of the extremity was highly suggestive of Buerger's disease with transmural inflammatory necrosis and thrombosis of the small vessels of the foot and distal tibial vessels, with no evidence of atherosclerosis or occlusion caused by microemboli or of occlusive disease of the proximal tibial vessels. The below-knee amputation healed well and the patient is ambulating with a prosthesis 15 months after amputation.

DISCUSSION

Embryology. The sciatic artery is a persistence of the embryologic axial artery, a continuation of the internal iliac artery as the major vascular supply to the lower limb bud in the early embryo (Fig. 5). The developmental anatomy of the lower limb bud was first reported by Senior[27] and has been extensively described by Arey.[28] After the 22 mm embryologic stage, the axial artery normally involutes and the femoral artery system develops as the major inflow source to the lower extremity. Remnants of the axial

Fig. 1. Selective angiogram of persistent sciatic artery (case 1) after thrombolytic infusion has successfully reopened proximal segment. Note persistent irregular surface caused by mural thrombus and calcified outer rim of aneurysm *(arrow)*. Distal sciatic artery appears free of disease and of normal size.

Fig. 2. Angiogram of patient in case 2 reveals both persistent sciatic artery *(large arrow)* and superficial femoral artery, which is hypoplastic in distal thigh *(small arrow)* and resembles arteriosclerotic occlusion.

artery normally persist in the adult as the proximal portions of the anterior and superior gluteal arteries, and distally as the popliteal and peroneal arteries.[28]

If there is failure in development of the femoral system or failure of regression of the axial artery, the axial artery may persist as a sciatic artery and function as the dominant supply to the lower extremity. The sciatic artery may be persistent bilaterally in some cases.[6,12,14,15,23] In the "completely" persistent sciatic artery, which constitutes approximately 63%[12] to 79%[24] of reported cases, the sciatic artery is the dominant supply to the lower extremity with continuity from the internal iliac artery into the popliteal artery.[12,15,24] In most cases of the complete variant, the external iliac and common femoral arteries are relatively normal in size but the superficial femoral artery is hypoplastic and terminates in the middle to lower thigh regions, with variable collateral communications with the popliteal artery. In the "incompletely" persistent sciatic artery, there is hypoplasia of the sciatic artery in the thigh, and the femoral system is the dominant arterial supply to the lower extremity, although there may be some collateral connections between the incompletely persistent sciatic artery and the popliteal artery.[3,10,15,24] There are reports of persistent sciatic arteries associated with hypoplastic or aplastic external iliac and femoral arteries[4,7,17] and of normal superficial femoral arteries that also supply the popliteal artery in addition to a completely persistent sciatic artery.[6,24] The rare occurrence of hypoplasia of both the femoral and sciatic arteries has been associated with significant underdevelopment or atrophy of the affected limb.[4]

Venous anomalies are infrequently observed with persistence of the sciatic artery. Bilateral persistence of both the sciatic artery and vein has been reported.[6] Congenital arteriovenous fistulas or malforma-

Fig. 3. Angiogram of patient in case 2 demonstrates persistent right sciatic artery *(arrow)* arising from hypogastric artery, with normal external iliac, common femoral, and proximal superficial femoral arteries.

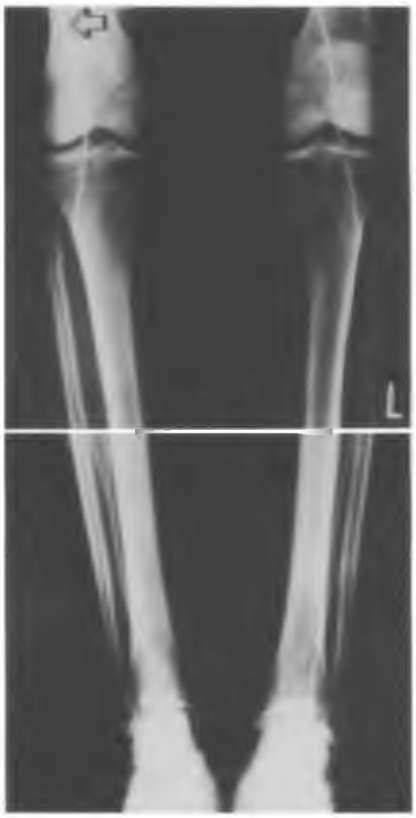

Fig. 4. Angiogram of patient in case 2 demonstrates that persistent right sciatic artery *(arrow)* enters popliteal fossa from lateral aspect and becomes popliteal artery. Proximal tibial and peroneal arteries appear normal but are occluded distally. Although there is some collateral filling of posterior tibial artery near ankle, no contrast could be demonstrated to enter pedal or digital vessels.

tions,[7,17] prominent extremity varicose veins,[25,26] and abdominal wall capillary hemangioma[25] have also been observed in association with a persistent sciatic artery.

Anatomy. When persistent into adulthood, the sciatic artery courses inferior to the gluteus maximus muscle, posterior to the greater trochanter of the femur, and along the posterior aspect of the adductor magnus muscle into the popliteal fossa where it becomes the popliteal artery.[3,8,11,18,23] The persistent sciatic artery may lie in close proximity to the sciatic nerve or in its sheath, or it may accompany the posterior cutaneous nerve.[2,11,12,14,24]

Pathology. "Arteriomegaly" of the persistent sciatic artery has been observed in many cases.[12,21] Aneurysmal dilatation of a persistent sciatic artery was first reported by Fagge[5] in 1864. The incidence of aneurysm formation in a persistent sciatic artery has been estimated to be as high as 44% in the comprehensive review by Williams et al.[24] The mean age at diagnosis of persistent sciatic arteries was 44 years, compared with the older mean age of 53 years at the discovery of a sciatic artery aneurysm.[24]

The etiologic basis of aneurysm formation in the persistent sciatic artery is unclear. Several early reports revealed a frequent association with a positive result on serologic test for syphilis,[3,8,9] but more recent reports have rarely noted this association, which suggests that the earlier reports were probably coincidental. It has been suggested that chronic trauma as a result of compression of the persistent sciatic artery against the sacrospinal ligament, piriform muscle, and hip, as well as frequent stretching during flexion of the hip joint, may also be a causative factor in aneurysm formation.[2,11,12,14,24] Others have suggested that there is congenital hypoplasia of the connective tissue components of the anomalous artery,[11,18,19,27] but there are few data to support this speculation. There is no evidence of a systemic connective tissue disorder.

Clinical findings. As noted previously, more than one half of reported cases of a persistent sciatic artery were discovered as incidental findings unrelated to aneurysm formation or other complica-

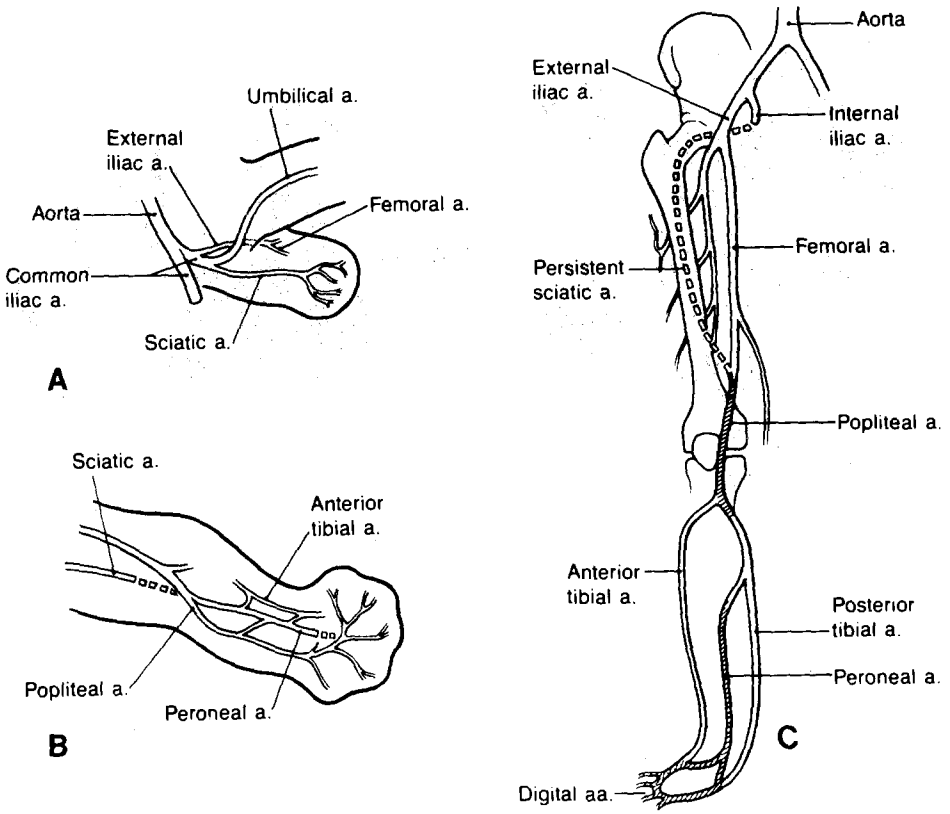

Fig. 5. Major arterial supply to lower limb bud in early development is sciatic (or axial) artery, continuation of internal iliac artery **(A)**. After 22 mm stage, femoral artery has usually developed into major artery, making connection with popliteal artery while most of the sciatic artery regresses **(B)**. After complete development **(C)**, remnants of sciatic artery normally persist as popliteal and peroneal arteries *(shaded regions)*; course of persistent sciatic artery is illustrated *(skipped segments)*.

tions.[14,24] The concurrence of persistent sciatic artery and arteriovenous malformations or varicose veins,[25,26] neurofibromatosis,[17] and ipsilateral limb hypertrophy[7,24,25] is infrequent and does not contribute to the clinical diagnosis.

If the femoral artery is severely hypoplastic or absent, the persistent sciatic artery may be suggested by the finding of palpable popliteal or pedal pulses in the presence of a markedly diminished[18] or absent[4] femoral pulse. In most cases, however, the femoral artery is sufficiently pulsatile in the groin to result in a normal pulse examination. If the superficial femoral artery is hypoplastic and the persistent sciatic artery insufficient to supply increased metabolic demands, claudication may occur.

Aneurysms of the persistent sciatic artery presented as pulsating masses in the buttocks in 31% of the cases reviewed by Williams et al.[24] Arterial insufficiency as a result of thrombosis of the aneurysm or distal embolization of mural thrombus from the aneurysm has been a common method of presentation.* Rupture has rarely been reported.[21,24] Other patients have been first seen with sciatica manifested by pain, numbness, or motor impairment as a result of compression of the sciatic nerve by the aneurysm at the level of the sciatic notch,[3,29] as was presumed to have occurred in our case 1 with onset 4 years before presentation with acute limb ischemia.

A clinically suspected sciatic artery aneurysm may be confirmed by ultrasonography, computed tomography, or magnetic resonance imaging. Angiography is essential to delineate that the aneurysm is related to a persistence of the sciatic artery and to define precisely the circulation of the distal extremity for possible reconstruction. It is important to distinguish sciatic from gluteal artery aneurysms, which can be simply ligated without ischemic complications, whereas aneurysms of the persistent sciatic artery often require vascular reconstruction to avoid severe ischemic complications.[3,8] The differential diagnosis

*References 2, 11, 12, 14-16, 18, 23, 24.

of a pulsatile buttock mass also includes abscess, enlarged bursae, sciatic hernia, granulomatous diseases, neoplasms, and congenital or acquired arteriovenous fistulas.[16,29]

Treatment. Incidentally discovered persistent sciatic arteries without aneurysm formation or other complications do not require interventional management,[26] as with our case 2. These patients should be monitored at regular intervals by physical examination and noninvasive imaging of the sciatic artery, with intervention recommended only if asymptomatic aneurysmal dilation is noted.

Aneurysms of the persistent sciatic artery should be occluded to prevent embolization or rupture. This has most commonly been accomplished by surgical ligation via a posterior approach as illustrated by Vimla, et al.[23] Percutaneous transcatheter embolization has been successful in occluding a persistent sciatic artery aneurysm by thrombosis.[12]

Ligation of the aneurysm may be the only treatment necessary in those patients with normal superficial femoral or adequate collateral perfusion of the lower extremity[3,9,10,15-17] or in patients with unsalvageable limbs or unreconstructible distal vessels.[11,14] In many instances, however, revascularization of the distal extremity has been necessary. This may be accomplished by interposition grafting from the hypogastric to the distal sciatic or popliteal artery[8] or by endaneurysmorrhaphy between undilated segments of the sciatic artery,[13,20,21,23] as in our case 1. No attempt should be made to excise the aneurysm and dissection should be minimized to avoid injury of the adjacent sciatic nerve.[20] Proximal vascular control may be achieved by retroperitoneal exposure of the hypogastric artery if it appears there will be insufficient normal-sized proximal artery for adequate control from the posterior exposure of the aneurysm.[3,8,16]

Many surgeons have chosen to revascularize the extremity by distal bypass grafts from the femoral artery when the femoral artery is adequate, because of their familiarity with this procedure and concerns about possible aneurysmal dilation of the residual segments of sciatic artery or occlusion of the residual artery and graft by external compression along its course.[2,14,15,19,22,25] However, to our knowledge there are no reports of such complications in the literature, and interposition grafting appears to be an acceptable method of revascularization.

Results of surgical treatment are dictated by the degree of ischemia before revascularization. Unfortunately, many cases have been discovered only after the onset of advanced ischemia, with amputation required as a primary or secondary treatment.[12,18]

To our knowledge, our case 1 represents the first report of thrombolytic therapy for acute ischemic complications related to thrombosis and emboli from an aneurysm of a persistent sciatic artery. Although there may be concerns that thrombolysis could precipitate embolization from mural thrombus in the aneurysm, there are significant potential benefits from reestablishing inflow and lysis of emboli and thrombus in the distal runoff vessels, which thus enhance the potential for successful surgical revascularization.

Our case 2 is the first report of a persistent sciatic artery associated with bilateral Buerger's disease, which was suggested by arteriography and confirmed by histologic examination of vessels in the amputated limb. There is no evidence that the distal occlusive process seen angiographically in both distal limbs was related to microemboli from the apparently undiseased unilateral persistent sciatic artery or from other sources.

The persistent sciatic artery is a rare but potentially significant vascular anomaly. The high incidence of aneurysm formation and limb-threatening ischemic complications must be recognized. With the application of modern vascular reconstructive techniques, these lesions can be managed successfully in many cases.

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