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Persistent Sciatic Artery: A Favorable Anatomic Variant in a Setting of Trauma

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Background: Persistent sciatic artery (PSA) is a rare congenital anomaly of the circulation of the lower limb that results from the persistence of an artery that normally regresses early in embryonic development. PSA is usually an incidental finding and is exceedingly rare to find bilaterally.

Case Report: We present the case of a rare presentation of PSA that resulted in a favorable outcome for a patient who sustained a gunshot wound to his midthigh and discuss the history, embryology, anatomy, classification schema, imaging evaluation, complications, diagnosis, and management of PSA.

Conclusion: PSAs are of doubtful clinical significance when found incidentally at imaging; however, individual patient symptoms, unique arterial anatomy, and the PSA classification best determine the appropriate treatment options.

Keywords: *Congenital abnormalities, femoral artery, lower extremity, persistent sciatic artery*

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INTRODUCTION

Persistent sciatic artery (PSA) is a rare congenital anomaly of the blood circulation in the lower limb that results from the persistence of an artery that normally regresses within the first 3 months of embryonic development. The incidence of PSA is estimated to be 0.025%-0.04%.[1] Bilateral PSAs are exceedingly rare and account for only 12%-32% of known cases.[1] It is right-sided in 50% of known cases, left-sided in 20%, and bilateral in $<$30% of known cases. Both sexes are equally affected.[2] Most patients with PSA are asymptomatic in their youth and present between the ages of 40-50 years old (mean age of 44 years) with claudication, aneurysm, and, rarely, sciatica.[2,3] Asymptomatic cases are found rarely and incidentally. PSA has been associated with an assortment of coexisting anomalies including Müllerian agenesis, arteriovenous fistulae, hypertrophy of the lower limb, varicose veins of an atypical pattern, and right retroesophageal subclavian artery. A genetic mutation or environmentally triggered vascular event in utero has been suggested to be the cause of these combined malformations.[4]

We present the case of a young male with bilateral complete PSAs.

CASE REPORT

A 19-year-old male presented to the emergency department after sustaining a gunshot wound to the left medial midthigh. The patient was brought to the hospital within an hour of his injury. The patient had no significant medical, surgical, or family history. On physical examination, the patient had an entrance wound to his left medial thigh, palpable pedal pulses and posterior tibial pulses, and full motor and sensory function throughout the affected lower extremity. Blood loss was minimal, estimated to be 25-50 cc in total. After the initial physical examination was performed and the patient's bleeding was stabilized in the trauma room, the patient was sent for computed tomography (CT) evaluation of his injuries.

CT examination was performed on a Philips 64-slice multislice scanner. Contrast-enhanced angiographic images from the mid-aorta through the lower extremities bilaterally were acquired, and sagittal, coronal, and 3-dimensional (3-D) reconstructed images were obtained.

Imaging revealed no evidence of injury to the pelvis, perineum, or right lower extremity. The arterial evaluation of the lower extremities showed the external iliac as it continued into the femoral artery along an expected course (Figure 1). Distal to the inguinal canal, however, the artery trifurcated in the proximal midthigh and quickly tapered into the muscular soft tissue beds (Figure 2). A bullet was lodged in the left midthigh (Figure 3) at the expected location of the left femoral artery. Additionally noted were the prominent but normal internal iliac arteries and patent bilateral PSAs that coursed adjacent to the sciatic nerve and formed the dominant circulation of both lower extremities. This pattern was consistent with bilateral complete PSAs. The PSAs supplied the popliteal arteries and formed normal-appearing posterior tibial arteries, anterior tibial

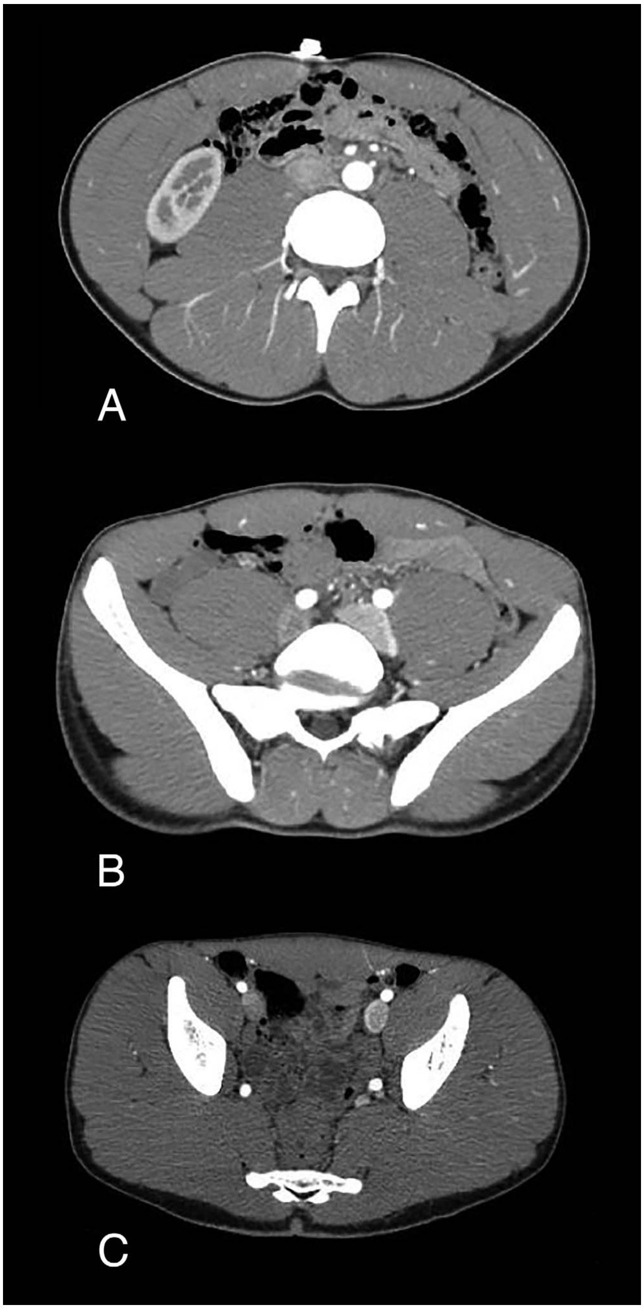

Figure 1. Axial computed tomography angiography images show (A) the level of the takeoff of the inferior mesenteric artery from the aorta; (B) normal bifurcation of the aorta into the common iliac arteries; and (C) the level of the internal and external iliac arteries. The internal iliac arteries are prominent but not aneurysmal.

arteries, peroneal arteries, and dorsalis pedis arteries bilaterally (Figure 4). The 3-D reconstructed anatomic views are shown in Figure 5.

Because the patient lacked a femoral artery as a result of this congenital anomaly, he avoided serious vascular injury and possible death from exsanguination resulting from the bullet's injury of the femoral artery in the medial thigh.

Because the patient had no evidence of vascular injury, pseudoaneurysm, or serious bleeding, the on-call surgery team decided to follow the patient medically in an outpatient setting, and the patient was discharged from the emergency department without removal of the bullet. There were no plans to remove the bullet at the time this article was written.

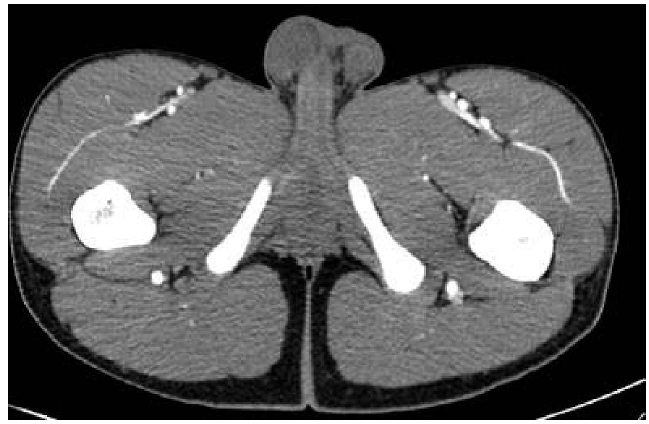

Figure 2. Axial computed tomography angiography image shows arterial anatomy with trifurcation of the femoral artery and prominence of a persistent sciatic artery just distal to the obturator foramen.

A review of the patient's social history showed that he was a high school track athlete and was able to return to his regular activities shortly after his trip to the emergency department without complication. The patient was followed clinically for symptoms related to his PSAs; he has had no symptoms.

DISCUSSION

Embryology, Anatomy, and Classification

Embryologically, the sciatic artery is a branch of the umbilical artery that forms a confluence of the capillary plexus in the limb bud[5]; this confluence consolidates to form the sciatic artery that serves as the primary arterial supply to the lower limbs during the 6- to 9-mm stage of the embryo. During the 10-mm stage, the femoral artery begins to develop as a continuation of the external iliac artery. By 12 mm, the femoral and deep femoral arteries are present.[4] The femoral artery enlarges and branches, eventually dominating arterial delivery to the lower limb. The 14-mm stage of the embryo is the only time during development that the lower limb receives dual blood supply from both the

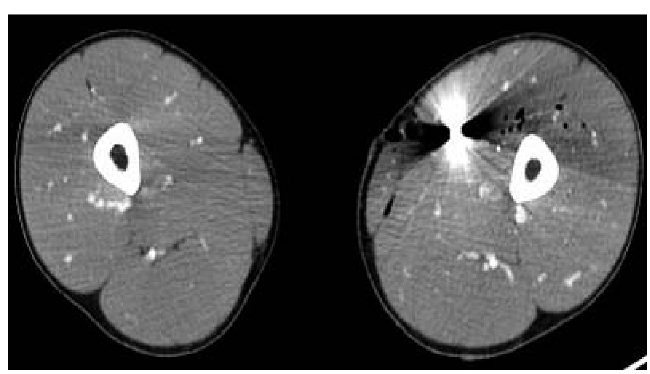

Figure 3. Axial computed tomography angiography image shows arterial anatomy in the midthigh with a bullet in the left medial thigh at the normal anatomic position of the femoral artery.

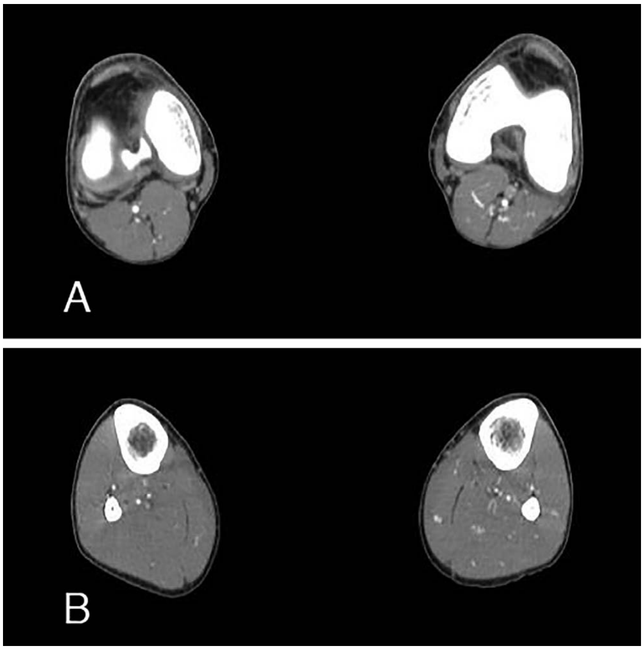

Figure 4. Axial computed tomography angiography images show (A) arterial anatomy at the popliteal that originates from the anomalous posterior circulation (the posterior persistent sciatic artery) bilaterally and (B) arterial anatomy at the distal popliteal with normal 3-vessel runoff into the anterior tibial, posterior tibial, and peroneal arteries.

sciatic and the femoral arteries. Normally, by the 22-mm embryologic stage, the sciatic artery has atrophied at its midpoint in the distal thigh.[5] Its proximal portion becomes the inferior and superior gluteal arteries, and the distal sciatic artery develops into the peroneal and popliteal arteries. The femoral artery continues to develop and establishes continuity with the popliteal artery, becoming the primary arterial supply of the distal lower limb.[6] Simultaneously, the umbilical artery develops into the internal iliac artery.[4] Several authors have suggested that the sciatic artery persists when the femoral arterial system is hypoplastic to deliver adequate blood flow to the developing limb.[4]

Anatomically, therefore, the PSA is essentially an anatomic extension of the internal iliac artery. The internal pudendal and superior gluteal arteries originate from the PSA within the pelvis. Subsequent to this branch point, the PSA courses posteriorly through the lower part of the sciatic foramen, beneath the piriformis musculature. At this level, the vessel is often accompanied by the posterior cutaneous nerve or sciatic nerve. Several authors of PSA cases have reported that the artery actually runs within the sciatic nerve sheath, although this finding is not universal.[4] At the proximal femur, the artery courses medially to the sciatic nerve. Occasionally, perforators from the profunda join with the PSA at this level. The PSA extends to the popliteal fossa via the dorsum of the adductor magnus, becoming contiguous with the popliteal artery. Subsequently, the PSA courses along the anterior aspect of the sciatic nerve and eventually terminates in the dorsal foot.[7] The PSA is typically subcentimeter in diameter, usually on the order of 5-9 mm. Although the sciatic artery and vein develop simultaneously along a similar course, it is extremely rare for the sciatic vein to persist along with the artery.[5]

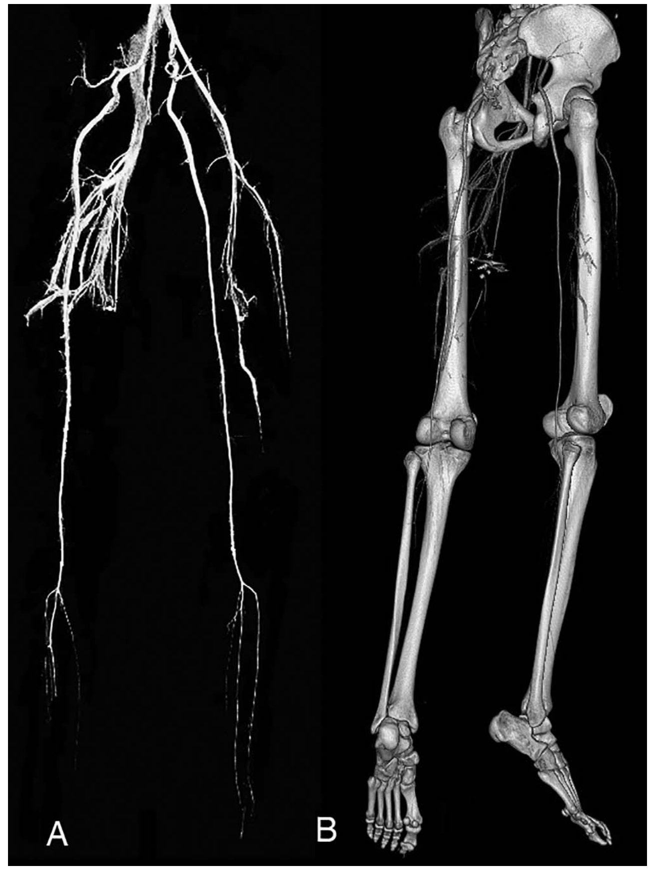

Figure 5. Three-dimensional reconstructed anatomic views from the front (A) and posterior-right (B) of the patient show the persistent sciatic artery (Type 2a) as it supplies the dominant circulation of the lower extremities. There is normal appearing 3-vessel runoff distally. Note that the bullet (with metallic artifact in the mid left thigh) overlies the expected position of the femoral artery that appears atrophic and does not connect to the popliteal. There is no evidence of acute arterial bleed.

A complete PSA is the most common variant and accounts for 70%-80% of recorded cases.[4] A complete PSA connects a large internal iliac artery to the popliteal artery. The femoral artery can be normal, incomplete, or absent.[1] In an incomplete PSA, the vessel is interrupted in its course from internal iliac to popliteal fossa but is connected to the popliteal fossa through collaterals.[8] In the case of an incomplete PSA, the femoral artery supplies a crucial fraction of arterial flow to the extremity and is continuous with the popliteal artery. The deep femoral artery can be absent or reduced.[1] Differentiating complete PSA from incomplete PSA is critical as the differences guide appropriate treatment options.[9]

PSA was first described in 1832 by Green during a postmortem dissection.[4] A classification system has been developed to best describe and catalog various PSA anomalies.[4] The classification scheme is outlined by incomplete or complete persistence of the sciatic artery and femoral development.

The PSA classification scheme describes 5 types (Figure 6). Type 1 is a complete PSA along with a normal femoral artery. Type 2 is a complete PSA with incomplete femoral development. In type 2a, the femoral artery is present but tapers and does not reach the popliteal artery. In type 2b, the femoral artery is completely absent, as in our case. Type 3 is an incomplete PSA in which only the proximal part of the sciatic artery remains, and the femoral arteries are fully developed. Type 4 is an incomplete PSA in which only the distal part of the artery remains, and the femoral arteries are fully developed. Type 5 PSA originates from the median sacral artery. Type 5a has a developed femoral artery, and type 5b has an underdeveloped femoral artery.[4]

Presentation

It is reasonable to believe that asymptomatic PSA cases are underreported because the asymptomatic individual does not seek symptom relief from healthcare professionals.[4] A PSA discovered as an incidental finding at imaging is of questionable significance. Occasionally, a PSA can be beneficial to the patient, as in this example of penetrating trauma in which a conventional lower extremity arterial anatomy would almost certainly have been affected.

As previously noted, patients with symptomatic PSA present at a mean age of 44 after an asymptomatic youth.[2,3] Presenting symptoms include buttock pain; pain and pallor of the lower extremity; claudication; poor capillary refill; cool extremities; black toes; and neurologic symptoms such as lower motor nerve weakness, sensory deficits, and pain in the sciatic nerve distribution.[2,5,8] On physical examination, Cowie sign—the absence of femoral pulse with the presence of distal pulses—is pathognomonic for PSA, although it is recognized in the minority of patients presenting with PSA.[2]

PSA aneurysm formation is the most frequent symptomatic pathology in patients with both complete and incomplete PSA, accounting for 43% of PSA presentations.[10] A PSA aneurysm manifests as a pulsatile mass located in the second third of the buttock. The artery's precarious position between the piriformis muscle and the sacrospinous ligament results in external compression of the PSA during hip flexion in the seated position that results in claudication while seated at rest.[8] A pulsatile PSA aneurysm is often painful as well.[2] A PSA aneurysm can grow large enough to compress the sciatic nerve, resulting in the aforementioned motor-sensory neuropathy in the ipsilateral limb. PSA aneurysms have also been reported to be associated with internal iliac artery aneurysms, limb shortening, and hemihypertrophy of the leg or pelvis.[8]

Several authors suggest that the PSA is prone to early atheromatous degeneration and aneurysm formation because of either an inherent collagen defect or hypoplasia of the connective tissue in the primitive arterial wall.[2,8] Some suggest that PSA aneurysms should be treated to protect against complications such as thromboembolism and rupture.[9]

The second most common presenting symptom of PSA is peripheral thromboembolism which may also be secondary to PSA aneurysm. However, even without PSA aneurysm formation, the lower limb may be at increased risk of ischemia because of the underdevelopment of the arterial supply.[11]

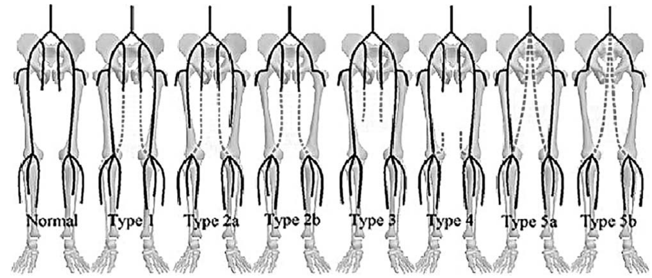

Figure 6. The persistent sciatic artery (PSA) classification describes 5 types of variant anatomy. Type 1 is a complete PSA with a normal femoral artery. Type 2 is a complete PSA with incomplete femoral artery development. In type 2a, the femoral artery is present but terminates in the midthigh. In type 2b, the femoral artery is completely absent. Type 3 is an incomplete PSA in which only the proximal part of the sciatic artery is present, and the femoral arteries are fully developed. Type 4 is incomplete PSA in which only the distal part of the artery is present, and the femoral arteries are fully developed. Type 5 PSA originates from the median sacral artery. Type 5a has a developed femoral artery, and 5b has an underdeveloped femoral artery.

Imaging, Diagnosis, and Management

A PSA can be diagnosed with Doppler ultrasound, CT angiography (CTA), or magnetic resonance angiography (MRA).[1] Digital subtraction angiography can also be used for PSA diagnosis and planning of its surgical repair. Characteristic PSA findings on angiography include enlargement of the internal iliac artery and/or a hypoplastic femoral artery. Popliteal and tibial vessels are often difficult to visualize on conventional angiography because of slow flow in the dilated PSA and may lead to a false impression of arterial occlusion. Lower limb ischemia secondary to PSA may also be misdiagnosed as a femoral artery occlusion if the catheter tip is placed directly into the external iliac artery or femoral artery. Such placement would reveal a tapered femoral artery.[5] The tapering effect of hypoplastic vessels may have the appearance of occlusion.[2] Identification of the PSA as the major inflow vessel is critical to avoid an inappropriate bypass of a hypoplastic femoral artery that appears occluded on angiography because of its underdevelopment.[6]

Several authors recommend using CTA because of these angiographic shortcomings.[1,5] CTA is a more comprehensive study that detects the artery's relationship to bony and muscular structures, the sciatic nerve, and femoral arteries and collaterals. CTA also provides information on the presence and size of an aneurysm, the presence or absence of a sciatic vein, the degree of intramural thrombosis, and PSA occlusion.[5] MRA provides similar information.[2] Characteristic findings on CTA are visualization of smooth tapering of the femoral artery and the distal stump of a PSA or an occluded vascular structure that had previously followed the sciatic nerve, both of which help to make the diagnosis of a total PSA occlusion.[5]

The role of sonographic color duplex scanning is principally for follow-up and monitoring of patients with PSA for occlusive events or atherosclerotic disease. Color duplex scanning has occasionally been used to identify the

PSA from a posterior approach; however, this method is not the test of choice for initial imaging.[2]

Treatment for patients with PSA depends on symptoms, the classification, and patient comorbidities. An asymptomatic PSA found incidentally does not require surgical treatment.[5] Long-term studies are still necessary to establish evidence-based guidelines, but annual Doppler duplex ultrasound surveillance is recommended because of the risk of aneurysm formation.[3] Oral anticoagulation that maintains an international normalized ratio between 2.5-3.5 and intravenous courses of prostaglandin E1 have been shown to improve claudication symptoms.[12] If medical therapy is chosen to treat claudication, ankle-brachial indices and duplex sonography are recommended every 3-6 months to follow treatment progress.[11] A CTA following oral anticoagulation is recommended to monitor treatment success and aneurysm size.[5]

Symptomatic PSA is usually addressed by surgery or radiologic intervention.[5] Several treatment options are available for a PSA aneurysm, the most common PSA pathology. A PSA aneurysm can be ligated, resected, embolized, or stented with an endovascular graft.[2] Aneurysm repair and arterial reconstruction have yielded acceptable intermediate and long-term results regardless of which method is chosen.[11]

Complete and incomplete PSA classifications determine the procedure of choice. A complete PSA provides the primary blood supply to the lower limb and must be preserved. Thus, revascularization should precede exclusion of an aneurysm. On the other hand, a limb with an incomplete PSA has reliable blood flow from the femoral arteries and collaterals, so exclusion of an aneurysm may not require revascularization; the PSA can be ligated or embolized without ischemic repercussions.[9,10] In fact, if an incomplete PSA has strong femoral arteries and collaterals or if the limb is not salvageable by revascularization, proximal and distal ligation of the incomplete PSA is recommended as thrombosis has been reported with proximal ligation alone.[8]

The gold standard treatment of PSA aneurysm with a complete PSA is revascularization along with exclusion of the PSA aneurysm because the lower extremity is dependent on the blood supply of the PSA.[6] Several options are available for revascularization, each with its own set of risks and benefits. Vascular reconstruction can be achieved by femoral-popliteal bypass, ilio-popliteal-transobturator bypass, or interposition bypass. End-to-end reconstruction after aneurysmectomy and resection is possible using autologous venous graft and/or prosthetic graft.[2] An interposition graft of the buttock or an ilio-femoro-popliteal graft can accomplish distal perfusion; however, placement of the graft may injure the sciatic nerve or compress the artery while seated. Additionally, the ilio-femoro-popliteal is a long graft that may become compressed by the adductor muscles and thrombosed.[10] A femoro-popliteal bypass is most commonly used if the femoral artery is sufficiently developed.[6,8,12] Follow-up with patients receiving these bypasses has shown that the femoral bypass and the PSA-route bypass have comparable postoperative results. The advantage of a PSA-route revascularization is that the PSA aneurysm repair and PSA-route bypass can be accomplished within a single surgical field with the patient in the prone position.[11]

Interposition grafting allows exclusion of the PSA aneurysm, restores blood flow, and preserves femoral vessels in case future surgery is required to maintain peripheral perfusion.[9] An obturator bypass has been recommended because the graft has a shorter route compared to the ilio-femoro-popliteal graft and thus may help prevent kinking and graft compression.[11] An obturator bypass involves ligation of the PSA and revascularization with interposition grafts between the internal iliac artery and the popliteal or distal sciatic artery.[8]

Several authors recommend endovascular embolization of the aneurysm sac as this procedure is minimally invasive and has a decreased risk of sciatic nerve damage compared to open surgical ligation.[8] The endovascular approach is not yet the gold standard despite the risk reduction because additional long-term follow-up studies are necessary; however, endograft recovery is a viable alternative to the open surgical approach, especially in high-risk patient populations.[9]

CONCLUSION

The PSA is a rare congenital vascular anomaly. Because of the high incidence of associated pathology, a PSA should be included in the differential diagnosis of lower limb ischemia or suspected aneurysm formation. PSAs are of doubtful clinical significance when found incidentally at imaging; however, individual patient symptoms, unique arterial anatomy, and classification best determine the appropriate treatment options.

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