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Case Report

Symptomatic Growth of a Thrombosed Persistent Sciatic Artery Aneurysm after Bypass and Distal Exclusion

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A 71-year-old woman presented with an enlarging mass in the right buttock, with pain and tingling sensation in sitting position. Five years ago, she was diagnosed with acute limb ischemia due to acute thrombosis of right persistent sciatic artery (PSA), and she underwent successful thromboembolectomy and femoro-tibioperoneal trunk bypass. Computed tomography angiography revealed a huge PSA aneurysm (PSAA). During the previous bypass, the distal popliteal artery was ligated just above the distal anastomosis to exclude the PSAA, whose proximal end was already thrombosed. However, PSAA has grown to cause compression symptoms, and the mechanism of aneurysm growth can be ascribed to type 1a or type 2 endoleak. In order to relieve the compression symptoms, aneurysm excision was performed without any injury to the sciatic nerve. A postoperative tingling sensation due to sciatic-nerve stimulation in the supine position resolved spontaneously one month after surgery.

Key Words: Aneurysm, Congenital abnormalities, Endoleak, Sciatica

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INTRODUCTION

Persistent sciatic artery (PSA) is a rare vascular anomaly that is prone to sequential changes such as vasculopathy, atherosclerosis, thromboembolic occlusion, and aneurysm formation. Any of these changes may lead to acute or critical limb ischemia of the lower extremity [1].

Aneurysm formation is reported in 15%-45% of PSA patients. Surgical excision or exclusion has been the treatment of choice for symptomatic PSA aneurysm (PSAA), but recently both endovascular coil embolization and stent-graft repair have been performed successfully, replacing the traditional procedures [2]. Herein, we present a patient with the rare finding of a growing PSAA after successful bypass and exclusion. The initial procedure was performed for PSAA thrombosis and distal embolization.

CASE

A 71-year-old woman complained of a tingling sensation in the right lower extremity and a palpable, non-pulsatile gluteal mass. The symptom was aggravated by sitting position, but she had no difficulty in walking. Her previous medical history included hypertension and deep vein thrombosis. Five years ago, she visited the emergency department due acute right leg pain and coldness, and was diagnosed with acute arterial occlusion. Her initial ankle-brachial index (ABI) was 0.49/1.09. Computed tomography angiography (CTA) revealed an acute thrombosis of PSA and embolic occlusion of right trifurcation in the calf. The

classification of the PSA was type 2a by Pillet-Gauffre classification [3] and Type IIIa by Ahn-Min classification [4].

She underwent successful femoro-tibioperoneal trunk bypass and ligation of the distal popliteal artery (Fig. 1). ABI was normalized to 1.04/1.13 after bypass surgery.

On physical examination, the arterial pulses in her right leg were all palpable. The palpable mass in the buttock was about 5 cm wide and 15 cm long, without tenderness or pulsation. Duplex ultrasonography showed triphasic waves of the femoral and crural arteries and total thrombotic occlusion of a sciatic artery aneurysm. CTA showed a PSAA that had enlarged from 2.5 cm at the time of initial surgery to 4.4 cm (Fig. 2). There was no flow detected through proximal artery and side braches into the aneurysm. To relieve the compressive symptoms of PSAA, surgical treatment was performed. The patient was placed in the

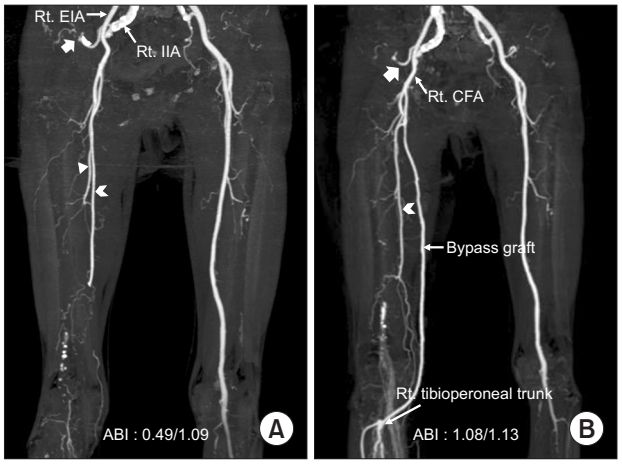

Fig. 1. Maximum intensity projection images of computed tomography angiography. (A) Preoperative image showing thrombotic occlusion of the PSA (arrow) which arose from the enlarged right IIA, a hypoplastic SFA (arrowhead), and DFA (triangle). There is no anomaly of the vascular structures on the left lower extremity. (B) Image after femoro-tibioperoneal trunk bypass showing good graft patency. PSA, persistent sciatic artery; EIA, external iliac artery; IIA, internal iliac artery; SFA, superficial femoral artery; DFA, deep femoral artery; CFA, common femoral artery.

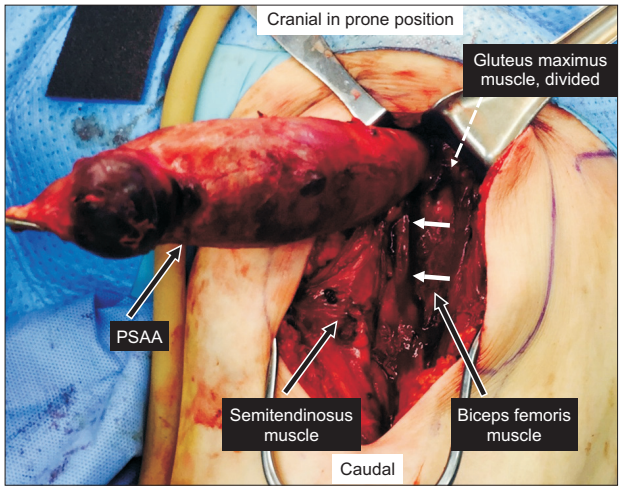

Fig. 3. Operative finding after PSAA excision in prone position. The sciatic nerve (white arrow) was located just below the anterior wall of PSAA. The PSA coursed between the biceps femoris muscle laterally and the semitendinosus muscle medially. The proximal part of PSAA could be exposed after division of lower part of the gluteus maximus. PSAA, persistent sciatic artery aneurysm; PSA, persistent sciatic artery.

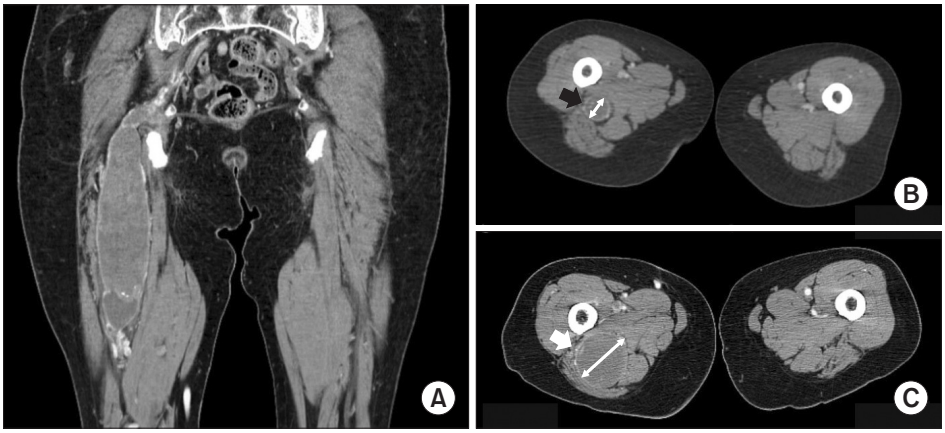

Fig. 2. Changes in images of computed tomography angiography during 5 years after bypass. (A) Coronal image at the second admission showing a huge persistent sciatic artery aneurysm (PSAA). No definite inflow to the PSAA was observed. (B) Axial image at the first admission. The PSAA (black arrow) diameter was 2.5 cm. (C) Axial image at the second admission. The PSAA (white arrow) diameter was 4.4 cm.

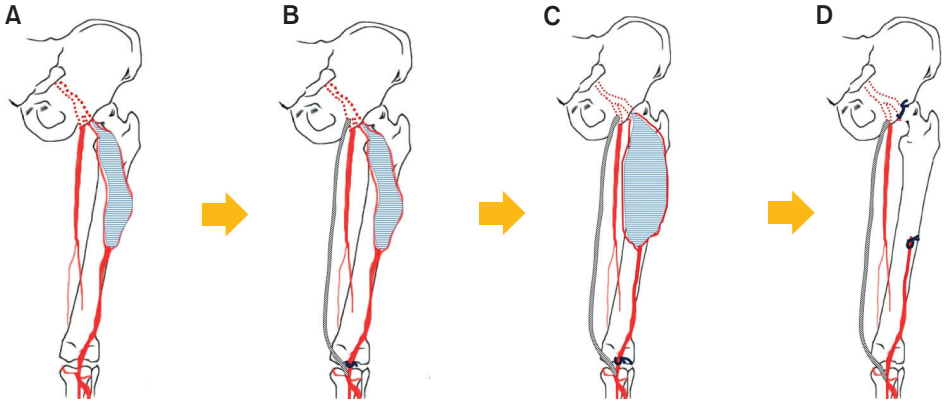

Fig. 4. Diagram of the overall surgical history of the patient in prone position. (A) Right PSAA (complete PSA with hypoplastic SFA) was diagnosed. (B) A right femoro-tibial bypass with distal popliteal artery ligation was performed due to thrombosed PSAA during the first admission. (C) The PSAA gradually enlarged to cause sciatic nerve compression symptoms after 5 years. (D) To relieve symptoms, PSAA was excised during the second admission. PSAA, persistent sciatic artery aneurysm; PSA, persistent sciatic artery; SFA, superficial femoral artery.

prone position, and a curvilinear incision was made on the right buttock and thigh over the PSAA. The aneurysm sac was exposed distally, and the underlying sciatic nerve was identified (Fig. 3). Initially, endoaneurysmal exclusion was planned to avoid injury to the sciatic nerve, but as the dissection plane was evident with little risk of nerve damage, the entire PSAA was excised. Multiple collateral vessels feeding the aneurysm were also ligated (Fig. 4). The patient complained of a postoperative tingling sensation while lying down, which resolved spontaneously after 1 month. Follow-up ABI after 9 months was 1.08/1.13.

DISCUSSION

An anomaly in the early embryonic development of the femoral-artery system causes PSA, a rare condition with a reported incidence of 0.03%-0.06% [1]. PSA was first reported by Green in 1832 [5]. Gauffre et al. [3] suggested a classification system based on the anatomy of the femoral artery (FA) and the sciatic artery. During fetal development, the umbilical artery emerges from the distal aorta; the primitive sciatic artery is made from the dorsal root of the umbilical artery when the embryo is 6 mm in length. When the embryo reaches 9 mm, the primitive sciatic artery provides the major blood supply to the lower extremity. The FA develops as a continuation of the external iliac artery, and the lower extremity receives a dual blood supply when the embryo is at 14 mm. By the time the embryo is 22mm in length, the primitive sciatic arteries become atrophic, and the FAs become larger as the major vessels supplying blood to the lower extremities.

Patients with PSA have either failed development of the FA system or a hypoplastic FA. PSA is classified into 5 types by Gauffre et al. [3]: type I, complete PSA and normal FA; type II, complete PSA and incomplete or hypoplastic FA (type IIa, superficial FA segment present [hypoplastic or no popliteal junction]; type IIb, superficial FA absent); type III, incomplete PSA (persistent superior segment) and normal FA; type IV, type III with an inferior PSA segment present; type V, PSA from the median sacral artery (type Va, superficial FA segment present; type Vb, superficial FA segment absent). Our patient initially had a type IIa PSA, composed of a complete PSA and a hypoplastic superficial FA. Although this classification may help to understand the embryologic perspective, they are not useful in making surgical decisions.

Recently, a new classification for PSA has been proposed by Ahn et al. [4]. It classifies into 4 types depending on completeness of PSA and femoral arteries and the presence of aneurysm. This new classification is simple and intuitive, which make it easy to plan surgical or endovascular treatment.

PSA is prone to pathologic transformation, including early atherosclerotic change, occlusive thromboembolism, and aneurysm formation. This rare vascular anomaly is associated with aneurysmal change in 15%-45% of patients. PSAA was first described by Fagge in 1864 [6], and bilateral PSAAs were observed in 25%-43% of patients with aneurysm development [2-4]. More than half of patients with PSAA have complications such as leg ischemia, persistent lower back or leg pain due to sciatic-nerve compression, or aneurysm rupture [7]. The treatment of PSAA depends on the type of PSA and on the presenting symptoms and signs. Surgical excision or exclusion has been the treatment of choice for symptomatic PSAA, but recently procedures using endovascular-coil embolization

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and stent-graft repair have been successful and have replaced the traditional treatment [1]. Surgical resection is preferable in a patient with a ruptured aneurysm who has either a type I or type III PSA. Exclusion can only be done safely in non-ruptured PSAAs. If there are not enough collateral circulations after PSA exclusion, additional bypass surgery is necessary.

Our patient initially presented with acute limb ischemia due to PSAA thrombosis and distal embolization. We performed thromboembolectomy of the tibial trifurcation and femoro-tibial bypass using the ipsilateral great saphenous vein. The distal popliteal artery was ligated just proximal to the distal anastomosis. Unfortunately, the aneurysm has subsequently enlarged, possibly due to mechanisms like type Ia or type II endoleak. Since no flow was detected, the proximal PSA was not ligated, which might have failed to block pressure conduction into the PSAA. This could be the cause of sac growth as in type Ia endoleak. Although type IIa endoleak was not detected on computed tomography, multiple branches to the PSAA might have caused the aneurysmal growth. To our knowledge, this is the first report of persistent sac growth in a thrombosed PSAA. We speculate that coil embolization or surgical exclusion of the proximal PSA during the initial operation might have prevented this growth.

Recent advances in endovascular therapy have provided an additional armamentarium for PSAA treatment. Endovascular coil embolization can be safely applied in cases with complete superficial femoral artery (SFA) [8]. Endovascular stent-graft repair is appropriate for complete PSAs and incomplete SFAs without symptoms of sciatic nerve compression. The advantages of stent grafting are the ability to shrink the aneurysm, the possibility for concurrent vascular reconstruction, the lack of risk to the sciatic nerve injury, and less invasiveness for high-risk surgical patients. However, there are technical limitations of endovascular stent-graft repair, and the durability and long-term results have not yet been reported [9].

Either a posterior or transgluteal approach may be used for surgical excision of a PSAA, although there is a risk of sciatic nerve injury. Batchelor and Vowden [10] recommended exclusion of the PSAA by proximal and distal ligation and suggested that the aneurysmal sac should be explored for additional feeder vessels. Drożdż et al. [11] reported that surgical excision of PSAA was a safe and effective method to avoid recurrence.

In conclusion, a distal exclusion of a thrombosed PSAA is not sufficient to prevent subsequent sac growth. Proximal exclusion by ligation or coil embolization may be necessary. Serial follow-up of the treated PSAA seems to be mandatory.

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